THE AURICULAR ARRHYTHMIAS



Figure 1. Photograph of right side of dog's heart showing anatomic relationship of right auricle and right ventricle. Note that dissection employed allows complete exposure of the right heart.

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CHARLES C THOMAS · PUBLISHER
Springfield · Illinois · U.S.A.

CHARLES C THOMAS • PUBLISHER
BANNERSTONE HOUSE
301-327 East Lawrence Avenue, Springfield, Illinois, U. S. A.

Published simultaneously in the British Commonwealth of Nations by
BLACKWELL SCIENTIFIC PUBLICATIONS, LTD., OXFORD, ENGLAND

Published simultaneously in Canada by
THE RYERSON PRESS, TORONTO

Printed in the United States of America

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Dedicated

TO OUR NEGLECTED FAMILIES WITHOUT WHOSE DEVOTION THIS WORK WOULD HAVE BEEN IMPOSSIBLE

I dined with Dr. M. Hall, and was indebted to him for one of the grandest spectacles that human eye can behold. I witnessed the circulation in the web of a frog, under a microscope of one-hundred magnifying power. I know not whether you have ever seen this or not; if not, you have no conception of its beauty. With what ideas does it fill the mind!

JAMES JACKSON, JR.
(In letter to his father, October 1, 1832)

Preface

Irregularities of cardiac rhythm have aroused the interest of scientists for many centuries. Not until the late nineteenth century, however, were techniques and apparatus developed which permitted accurate analysis and classification of the various types of arrhythmia. During the following decades the auricular arrythmias – premature systole, tachycardia, flutter and fibrillation – were subjected to extensive experimental study and diverse theories concerning their mechanisms were proposed. This series of investigations was climaxed by the work of Sir Thomas Lewis Despite the limitations imposed by the relatively crude methods available, Lewis's studies were remarkably thorough. Indeed, his observations on the auricular arrhythmias were considered so authoritative, and his circus movement theory of auricular flutter and fibrillation was so widely accepted, that cardiologic research largely turned from the auricles to the ventricles.

The fact that few investigations of the auricular arrhythmias have been conducted during the past 30 years is both fortunate and unfortunate Recent progress in the understanding and treatment of the more serious cardiovascular disorders, such as coronary disease, might have been delayed had the attention of investigators remained on the auricles. On the other hand, despite the thoroughness of Lewis's study and the occasional contributions of subsequent workers, few questions concerning the nature of the auricular arrhythmias have been definitely answered. Why these workers have been unable to establish final conclusions is apparent from a review of the literature. Because of limitations of technique, no one has advanced convincing evidence, based on direct observation, of precisely what takes place in the auricles of man or of the experimental animal during normal sinus rhythm or any of the auricular arrhythmias.

HISTORY OF THE STUDY

Just as technical advances provided the impetus for initial investigations of the auricular arrhythmias, so the availability of improved equipment encouraged us to hope that the present study of these disturbances might prove fruitful. The majority of the experiments performed in the course of the study were devised by previous workers. But the methods of observation used by the workers were too indirect and too insensitive to yield conclusive results. Only with the recent development of two new instruments – the high-speed cinematograph and the cathode-ray oscillograph – has it become possible to determine the precise nature of the mechanical and electrical events which occur in the auricles.

In 1947 we began our examination of the auricles with the high-speed cinematograph. By means of this instrument, the course of the auricular contraction wave was clearly visualized for the first time. Even during preliminary studies of normal sinus rhythm, the advantages of the cinematograph over previously available methods of observation were confirmed. Aspects of the normal contraction of the auricles which hitherto had been subjects of speculation were distinctly visualized; indeed, many features demonstrated in the motion pictures were contradictory to prevailing concepts. Since previous methods had failed to yield an accurate impression of the simplest rhythm, it is scarcely surprising that no definitive conclusions had been reached concerning the much more complex events occurring in the auricular arrhyth-

mias. In subsequent months the motion of the auricles during each of the experimentally produced arrhythmias was recorded by means of the high-speed cinematograph. In our opinion, these records constitute the most direct and most convincing evidence obtained in the present or previous studies of the auricles (during normal rhythm or any of the auricular arrhythmias). Unlike electrocardiograms which may be subject to error of interpretation, the motion picture provides an unequivocal record of events exactly as they occur in the auricle. When films are recorded under high magnification and projected on a large screen at slow speeds, even the minutest details of auricular motion are revealed. Unfortunately, despite careful selection and diagramming, the still photographs reproduced throughout this monograph give the reader only a poor conception of the fascinating movements of the auricle seen in the pictures.

A second largely unexploited method of observation used extensively in our studies is the dual-beam cathode-ray oscillograph. This electronic apparatus provides a graphic representation of the electrical activity of the auricle over 1000 times the size of the standard electrocardiogram. The oscillograph proved most valuable for the examination of the rapid activity of the fibrillating auricle. Details of the other arrhythmias which were not recorded with standard electrocardiographic equipment also were revealed in the oscillogram.

Of the experimental observations reported in this monograph, some 30 per cent were made with the high-speed cinematograph and the remainder with the multiple channel electrocardiograph and the cathode-ray oscillograph. Direct determination of the electrical and mechanical activity of the auricles was further facilitated by a new operative technique which permits complete exposure of both left and right auricles.

Thus, the original material reported in the following pages represents a combination of old experiments with new methods of observation. When the same experiments were recorded on previously available apparatus, the results were confusing and inconclusive. It is not surprising that the more direct and more sensitive methods now available should yield what appears to be to us definitive proof concerning the mechanism of the auricular arrhythmias.

The entire study consumed a period of approximately four years. Originally, only a thorough cinematographic examination of the motion of the auricles in experimental animals was planned. By the end of 1947, films had been recorded which demonstrated to our satisfaction the mechanism of the auricular arrhythmias as well as several previously unknown features of normal sinus rhythm. These films were presented during the winter of 1947-48 before various national medical meetings. Considering the newness of the cinematographic technique, coupled with the fact that the observations were contradictory to firmly entrenched theories, it is understandable that the conclusions were received with general skepticism.*

In order to obtain evidence corroborating the cinematographs, the mechanism of the auricular arrhythmias was further investigated with more conventional electrocardiographic techniques. During this phase of the investigation, covering the past three years, Lewis's experiments were repeated and extended. The course of the cardiac impulse was traced by means of electrocardiographically and oscillographically recorded direct auricular leads from experimental animals and esophageal leads from man. Our studies of the clinical arrhythmias were greatly facilitated by the generosity of colleagues throughout the country who provided us with rare and interesting tracings, many of which

* Such skepticism is understandable The effect of Lewis's work on the general opinion concerning the mechanism of the auricular arrhythmias is witnessed in a review of pertinent literature Early in the course of the present investigation a search was made of over 30 standard up-to-date textbooks in the fields of medicine, cardiology, physiology and pharmacology. In the majority of these textbooks the existence of a circus movement in auricular flutter and fibrillation is stated as a fact occasionally, circus movement is described as a theory, rarely, it is presented together with alternative theories. The therapeutic effect of drugs used in the treatment of auricular flutter and fibrillation generally is ascribed to their ability to abolish the "excitable gap" on the hypothetical circus pathway

are worthy of publication in medical journals. Finally, as the opportunity arose, cinematographic records of experimental and spontaneous auricular arrhythmias in human subjects were made simultaneously with indirect and esophageal lead electrocardiograms. Five basic facts emerged from this phase of the study: First, the electrocardiographic and oscillographic observations confirmed the earlier cinematographic observations. Second, in every instance in which observations of spontaneous or experimentally produced arrhythmias in man could be compared with observations made in experimental animals, the principles true in dogs were found to apply also to humans Third, observations on the mechanism of the auricular arrhythmias in man consistently could be made more easily than corresponding observations in the experimental animal, and without harm to the subject. Needless to say, the elucidation of the disturbance is far more significant in man than in the animal Fourth, the current method of interpreting electrocardiograms of the auricular arrhythmias is unsound and confusing. An alternative method has been devised. Fifth, the effect of quinidine and digitalis on the auricles may be explained without reference to the circus movement theory Whereas treatment of the arrhythmias currently is largely empirical, the demonstration of the mechanism of the disturbances and certain aspects of the pharmacologic actions of anti-arrhythmic drugs provides a more rational and understandable basis for therapy

ORGANIZATION OF THE MONOGRAPH

The material presented in this monograph is divided into 18 chapters and an appendix. Preliminary drafts of the text included only original experimental observations together with a review of earlier investigations of the mechanism of the auricular arrhythmias. As the study progressed, the comparability of the arrhythmias in man with those in the experimental animal became apparent and the inclusion of three types of clinical material was considered worth while First, clinical counterparts of experimental observations were added to illustrate the relationship between events observed in the laboratory and those known to occur in patients. An entire chapter has been devoted to the application of a new method of interpreting electrocardiograms of the auricular arrhythmias. Second, general clinical data compiled from an extensive review of the literature has been included at the beginning of the discussion of the mechanism of each arrhythmia. Third, a chapter summarizing current knowledge concerning the practical therapy of the auricular arrhythmias has been added. Since we have found that patients with the same arrhythmia may require different management, the therapy outlined in this chapter has been designed for application to specific situations which the clinician is likely to encounter rather than for use in a particular arrhythmia.

The Appendix consists of a detailed description of the various types of equipment and techniques used in the study. Some of these details may not be of interest to the general reader. Other investigators, particularly those who intend to repeat or extend the observation, may find the information in the appendix less complete than is necessary for their purposes. Further information concerning the methods used in the study will be supplied upon request.

Insofar as possible, the subject matter throughout the monograph is presented in a simple and readily understandable manner. Nevertheless, a certain minimum of highly technical material has been included for the benefit of specialists (who otherwise might find the discussions incomplete). The chapter on the pharmacology of quinidine and digitalis (Chapter XVI) includes considerable detail which is not of clinical interest and is not essential to an understanding of other portions of the text. Pharmacologists and internists particularly concerned with pharmacologic problems may consider this chapter worthy of study. The general reader is advised to disregard the body of the chapter and to read only the concluding summary which will provide an adequate picture of the clinical effects of the drugs.

From the performance of the first experiment to the completion of the final manuscript, the preparation of the material presented in this monograph has been a team job. Throughout the study the willing efforts of members of our staff were supplemented by generous contributions of time and material on the part of cooperative colleagues. Finally, every effort has been made to present an accurate picture of previous investigations of the auricular arrhythmias. The number of valid observations made in the past, despite technical limitations, is indeed remarkable. It is a pleasure to acknowledge that many techniques introduced by previous workers have been of inestimable value in our studies.

When Lewis finished his study of the mechanism of the auricular arrhythmias, the impression prevailed that the subject was largely understood. Thirty years later, it was apparent that much remained to be learned. It is equally apparent that the present study leaves many questions still unanswered. The action of drugs, especially digitalis, on the auricles; further cinematographic study of the human auricles, particularly the left auricle; nature of the auriculoventricular conduction systems, certain aspects of the nature of auricular fibrillation – these and many other problems await elucidation. In the future, when present methods are more fully exploited and new methods developed, others will discover fascinating facts about the auricles. Meanwhile, it is hoped that the data contained in the following pages will prove of use to medical students, clinicians and workers in related fields, and as a basis for further research by other investigators.

In conclusion, the authors desire to thank the many individuals who aided in the preparation of this monograph Acknowledgment is due numerous colleagues and friends who offered us the opportunity of studying and utilizing their clinical material. We are especially grateful to Dr George C Griffith, who kindly permitted us to use his electrocardiographic files, including valuable heart catheterization electrocardiograms, and who made available a number of his patients for special electrocardiographic studies. We are indebted to Dr. Mervin J. Goldman and M. Sokolow who provided the electrocardiographic material for an analysis of the site of origin of human auricular fibrillation; to Dr. D. M. Green for the use of his electrocardiographic tracings made during cardiac catheterization; Dr. Charles D. Enselberg who permitted us to to analyze and reproduce a number of esophageal lead electrocardiograms from patients with auricular tachycardia and flutter; to Dr. Lewis T. Bullock, who kindly allowed us to use his records of auricular tachycardia in infants; and to Dr. Howard B. Burchell who permitted us to publish phonocardiograms and pulse tracings from patients with auricular flutter.

It is impossible to make specific acknowledgment to the host of individuals who developed the high-speed cinematographic technique upon which much of this study is based. Our special thanks go to John R. Bishop and John H. Waddell for invaluable advice, and to Dale Gillette, S. F. Sanford, Robert Moreno, and Max Worley, who gave so freely of their time and effort.

We are grateful to Drs. Clarence M. Agress, Stephen R. Elek, Eugene B. Levine, Lawrence G. Stuppy and Travis Winsor for their interpretations of electrocardiographic tracings

Acknowledgment is due our extremely qualified colleagues who read and criticized preliminary drafts of the text, Dr John Martin Askey, Harry Goldblatt, John S. Lawrence, John J Sampson, and M Sokolow. Their criticisms and suggestions have been most helpful

We are indebted to Drs. Paul D. White, J. J Silverman and O. M. Race, Franz M. Groedel, and their respective publishers, for allowing us to reproduce various illustrations, to Dr Frank N. Wilson for his wise counsel, to Norma Lazar, Isobel Skoborg, Louis Fields, Elsa Leiter and Arthur Riesenfeld for technical assistance, and to Mrs. Mary Mayo Cosby for the statistical analysis in Chapter XIV. Mr M. Rappaport of the Sanborn Corporation has been of great assistance in checking the accuracy of our oscillographic equipment. We wish to thank the Technicon Cardiograph Corporation for the use of

their cardiograph and the Sanborn Corporation for the use of their Dualbeam Electrocardiograph

This work was made possible by the financial support of the Beaumont Trust Fund (founded by Louis D Beaumont). Our heartfelt thanks to our friends Morton May, N. Dauby, and N. Loeser, the Trustees of the Beaumont Fund, and to Mrs. Margaret Mayer, J. M. Schenk, L. Spitz, Ben Jacks, and Mrs. Tom May, without whose generous financial help this study could not have been undertaken.

Our publisher, Charles C Thomas and his son, Payne, have been most helpful and cooperative The excellence of our color plates is due to the craftsmanship of Mr Homer Alexander and the helpful advice and cooperation of Dr. H. T. Kalmus and Mr. Jack Clark.

The authors wish to express their deep appreciation to Mrs. Howard Finston, for upon her rested the burden of editing the manuscript down to the last comma, helping to organize the material into a logical sequence, and revising the authors' writing into clear, concise and understandable English. She is responsible more than anyone else for the style and appearance of the material in the text.

For complete unstinted cooperation of Dr. Harry Goldblatt, the Director of the Institute for Medical Research, we are grateful beyond measure.

The Authors

Contents

THE AURICULAR ARRHYTHMIAS

CHAPTER 1

The Normal Auricles

ANATOMIC RELATIONSHIPS

The Right Auricle: The right auricle is situated cephalad to the right ventricle directly over the tricuspid orifice. It is attached to the ventricle along the auriculo-ventricular groove; its medial wall, formed by the interauricular septum, separates it from the left auricle. The auricle itself is divided into two parts, the body and the appendix. On its dorsal aspect, the auricular body receives the superior and inferior venae cavae, ventrally, it opens into the tricuspid orifice

In the dog or human, when the heart is photographed from its right side, with the subject supine and tilted to the left, the right auricle is readily visible (Figure 1, Frontispiece). It appears as a bluish-red, elongate structure, framed on all but its dorsal border by the right ventricle. The appendix extends to the left as an out-pocketing from the body. The superior vena cava penetrates the dorsal border of the body obliquely from the left, the inferior vena cava enters at a more obtuse angle from the right [illegible]

[illegible] which lies in the auriculo-ventricular groove. Distal to the halo of fat is the dark-red right ventricle

The Left Auricle: The left auricle lies cephalad to the left ventricle immediately over the mitral orifice. Like the right auricle, it is divided into two parts, the body and the slightly smaller appendix. On its dorsal surface the body receives the pulmonary veins, approximately eight in number Because it is anchored to these veins and to the left ventricle, the body is almost immobile. The appendix is fan shaped in outline and is slightly constricted at its junction with the body; it is longer than its counterpart on the right (Figure 2).

In the dog or human when the left side of the heart is photographed with the subject supine, the left auricular appendix occupies the center of the picture, nestled in a hollow between the pulmonary artery and left ventricle. Its color is a brilliant red, in contrast to the bluish-red of the right appendix. Its surface is wrinkled; in many instances its margin is indented, presenting a serrated appearance. Below and to the left of the base of the appendix, the posterior half of the body may be seen. Its surface is smooth and it is dark bluish-red in color. Several pulmonary veins enter the body at its lower borders. Surrounding the base of the auricle is the yellow-white fat lying in the auriculo-ventricular groove.

In both auricles, the interior surface of the body is smooth; that of the appendix is ridged by the musculi pectinati.

The Interauricular Septum: The interauricular septum cannot be seen on films of the intact heart; it extends as a sheet between the right and left auricles, effectively separating these chambers (Figure 3).

The septum is composed of cardiac muscle fibers with a groundwork of connective tissue in about the same proportion as in the remainder of the auricular musculature.

No division separates the structures in the septum from those in the adjacent auricular areas. The superficial layer of muscle [illegible]

[illegible] elements of the right and left auricles is provided by the muscle fibers of the interauricular septum. The endocardium of the septum is also continuous with that of both

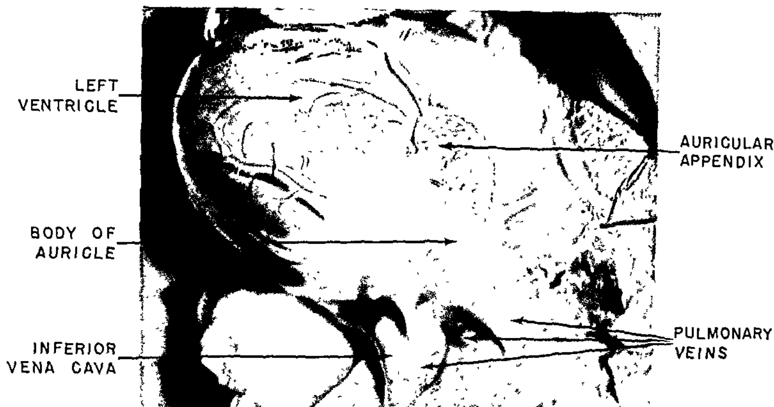


Figure 2 Photograph of left side of dog's heart showing anatomic relationship of left auricle and left ventricle Note body of left auricle is bound down by pulmonary veins and left ventricle. Appendix is most easily accessible part of left auricle

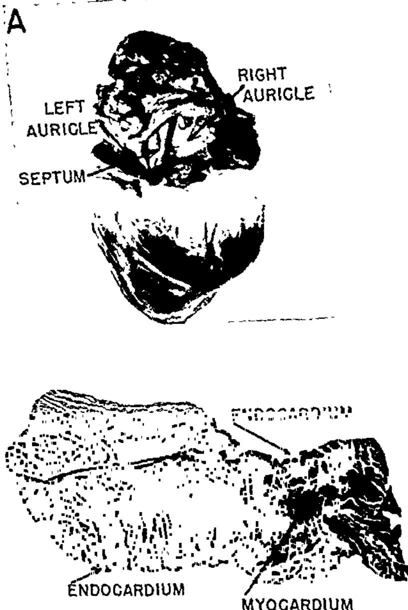


Figure 3 (A) Posterior aspect of heart showing gross anatomic relationship of interauricular septum and the two auricles Note separation of right and left auricular chambers by septum

(B) Photomicrograph (10 x) of interauricular septum, Wide band of myocardium, contiguous at each end with myocardium of auricles (not shown), is lined on each surface with endocardium

auricular chambers. *Clearly the septum is structurally an integral part of the auricles.*

The Conduction System: The structure and function of the auricular conduction system, either in man or in dog, is not agreed upon.[130] In 1907, Keith and Flack[303] described a neuromuscular bundle (the sino-auricular node) in the auricular myocardium near the opening of the superior vena cava, similar in structure to the auriculo-ventricular node previously described by Tawara;[601] this site was considered the source of the cardiac impulse. No definite anatomic conduction pathway through the auricle has been demonstrated in man or dog.

The following description of the anatomy and physiology of the sino-auricular node in man represents the majority viewpoint.

The sino-auricular node is a club-shaped structure which lies in the wall of the right auricle at the entrance of the superior vena cava. This location is marked by a slight ridge known as the taenia terminalis. The node is 2 to 2.5 centimeters in length and 2 to 5 millimeters in diameter. An elongated, conical head and a narrower tail may be distinguished. There appear to be transitional nodal-muscular fibers extending from the head and tail of the node out into the auricular musculature proper. Ac-

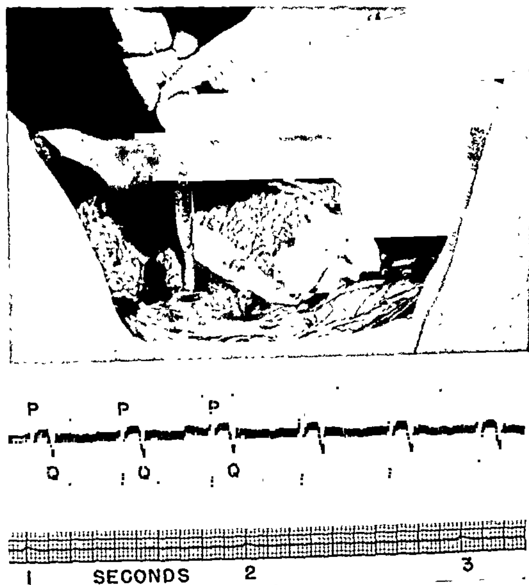


Figure 4 Upper photograph demonstrates single non-polarizable electrode being held lightly against the right auricular wall. Lower electrocardiogram demonstrates tracing made directly from surface of the auricle Note the characteristic intrinsic deflections of the P wave.

cording to some investigators, anatomic connections exist between these transitional cells and ordinary auricular muscle fibers.[649]

The impulse is believed to pass from the sino-auricular node to the auriculo-ventricular node via the auricular musculature. No convincing evidence has been advanced to confirm the existence of a special conduction bundle connecting the two nodes.

ELECTROGRAPHIC CONSIDERATIONS

In the present study the electrical events associated with the heart beat in experimental animals and in man was recorded on the standard electrocardiograph and the cathode-ray oscillograph The oscillograph represents an improvement over standard electrocardiographic equipment. It was used most extensively in studying auricular fibrillation and other rapid rate rhythms, however, it also proved useful in demonstrating minute details of the electrical activity in the normal auricle which were indistinct or imperceptible in the standard electrocardiogram

The Electrocardiograph: The electrical events of the auricular cardiac cycle consist of waves of depolarization (excitation or accession) and repolarization (regression). In other words, the nature of electrical activity of the auricles is the same as that occurring in the ventricles The electrical events of these two phases of auricular activity are not as striking nor as widely understood as their counterparts in the ventricles

The P wave is the wave of depolarization or the first phase of auricular activity.[2] In limb leads and precordial leads in normal hearts, the P wave is usually monophasic or diphasic. In esophageal leads at auricular levels and in direct auricular leads, the P wave is polyphasic* and of greater amplitude than in the limb leads. In each esophageal and direct auricular lead (Figure 4) a sharply defined intrinsic deflection is inscribed as the impulse passes beneath the electrode. According to present electrocardiographic concepts, the inscription of the P wave is due to the depolarization of the muscle strip (Figure 5). For the purpose of simplicity, in this monograph the term "P wave" is used to embrace the entire depolarization process in normal sinus rhythm; "P' wave" is used to describe the similar process in the arrhythmias.

The occurrence of a wave of auricular repolarization following the wave of depolarization was established in 1906; it was named the Ta wave by Hering[270, 271, 272] (1908). Macleod[419, 420] demonstrated in the frog's heart that the wave of repolarization starts before the wave of depolarization is terminated. The Ta wave is inscribed at a period normally occupied by the ventricular QRS complex and usually is masked by it, except when complete or partial auriculo-ventricular block is present (Figure 6). Clinically, Sprague and White[675] studied standard leads from 37 patients with complete heart block; the presence of Ta waves was shown definitely in 18 instances and inconclusively in eight others. As a rule, the normal Ta wave is deflected in a direction opposite that of the P wave and is *small* (rarely greater than 0.2 millivolt). This has also been clearly demonstrated in esophageal leads[73] and intracardiac leads,[31, 268, 568] in both of which it is often more distinct than in limb leads.

That the P wave is the wave of depolarization and the Ta wave is the wave of repolarization may be shown by means of simultaneously recorded electrocardiograms in both man and animals In man, esophageal electrocardiograms at various auricular levels have been taken s[illegible] leads. T[illegible] occur d[illegible] P wave, never during the Ta wave. The same experiment has been done repeatedly with direct auricular leads from various parts of the auricles in dogs; here too, the intrinsic

* The parts of the polyphasic P wave have been given various names by workers in the past Brown[illegible] called the deflections of the P wave in the esophageal electrocardiograms s, a, e, ι, o, and υ, Battro and Bidoggia[illegible] followed this terminology Hecht,[illegible] on the other hand, has adopted the standard nomenclature of the QRS complex for the P wave and calls the various parts P_Q, P_{QS}, P_{QRS}, P_R, and P_S He considers that this nomenclature facilitates description of the various complexes obtained from intracardiac and esophageal leads

human subjects during surgical procedures on the heart and lungs. This method of observation yielded direct and conclusive evidence regarding many features of the heart beat which hitherto had been subjects of speculation.

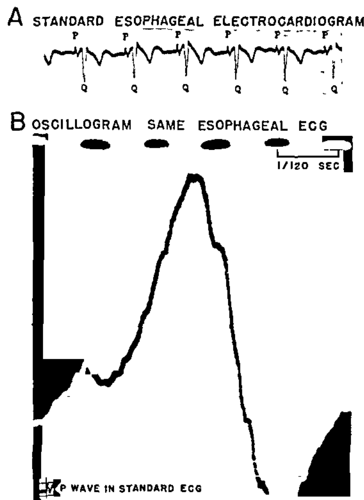


Figure 7. (A) Unipolar, esophageal lead from auricular level, normal male Standard electrocardiogram.

(B) Same subject. Auricular complex recorded and magnified by means of oscillograph Camera speed increased from 25 mm. per second to 381 mm per second Amplitude increased 32 times Insert at lower left hand corner of B shows the same P wave obtained with standard electrocardiogram equipment.

Observation 1: Site of Origin of the Normal Contraction Wave

As stated above, in 1907 Keith and Flack [303] suggested that the cardiac pacemaker might be located in the mass of specialized tissue found in the wall of the auricle near the superior vena cava. The suggestion was based upon the relatively large size of this mass and its intimate association with the many nerve fibers supplying the heart. However, up to the present time, no general agreement has been reached as to the *exact* site of origin of the cardiac impulse.

MacWilliam in 1887,[422] utilizing the known effects of temperature changes on the functional activities of tissues, found that the "application of slight heat locally to the terminal part of the vena cava superior gives a marked acceleration in the rhythm of the whole heart." Conversely in 1910 Flack [196] reported that a localized cooling agent slowed the rate of the heart only if it was applied to the region of the sino-auricular node. Gantner and Zahn in 1911 [214] likewise found that cooling of the sinus node slowed the rate of the heart beat; in addition, they noted that the farther the point of cooling was removed from the node, the less marked was its effect.

Lewis [337, 382, 385] and others,[50, 54, 55, 56, 99, 169, 171, 413, 414, 459, 679] investigated this problem by a variety of methods, including destruction of the sino-auricular node and determination of the point of primary negativity in the auricle. Lewis [385] found "abundant and conclusive evidence that the excitation wave begins in or near the head of the sino-auricular node." Eyster and Meek [177, 178, 179, 180, 182, 183, 184] confirmed many of Lewis' observations. On the other hand, Rijlant and Geraudel, according to Wiggers,[648] held the opinion that the normal beat actually arose from a restricted locus (1 millimeter in diameter) superior to the sino-auricular node Wiggers [648] further stated that "while we may continue to regard the region of the sino-auricular node as the normal pacemaker, it is necessary to broaden our conception to include the possible origin of the impulse in adjacent tissue." To add further confusion, Glomset and Glomset [231] and others were unable to obtain any histologic evidence of the existence of a separate specilized sino-auricular node. Nonidez,[469] however, found histologic evidence of a node in hearts of puppies and in the monkey

From electrocardiograms taken directly from the region of the sino-auricular node, the site of origin of the cardiac impulse cannot be determined. Deflections from the node, and those from points adjacent to it, resemble each other too closely. Similarly indeterminate are the

relatively crude cooling experiments.

By direct visualization of the *dog's right auricle* on high speed cinematographs, the *exact site* on the auricular surface of initiation of the *contraction* wave has been clearly seen for the first time. Motion pictures in 25 animals consistently showed that the *contraction wave starts in a region on the sulcus terminalis (corresponding to the taenia terminalis) at a distance approximately 1 centimeter to the right of the junction of the right auricular appendix with the superior vena cava* This locus is nearly equidistant from the caudal and cephalic extremities of the auricle (Figure 8).

If the anatomy of the sino-auricular node in the dog corresponds to that usually described for man, the locus of initiation of the auricular contraction wave in the dog is about *halfway* between the head and tail of the node, perhaps somewhat closer to the tail. Contrary to the view now held by most workers, the wave does not originate in the head, as revealed by the films, the location of the head is approximately 1 centimeter to the left of the region in which the contraction wave is seen to arise.

OBSERVATION 2· NORMAL CONTRACTION OF THE RIGHT AURICLE

In colored motion pictures taken at 1000 or 2000 frames per second, the first manifestation of auricular systole observed on the screen is a *wrinkling of the* body of the right auricle near the *tail* of the sino-auricular node (site of initiation of contraction wave). From this locus a wave of contraction spreads rapidly over the auricle in an ever-widening half-circle, completely engulfs that chamber, and leaves it contracted in all its dimensions. When the wave reaches the periphery of the auricle, *maximum* systole is attained. As the contraction subsides, relaxation into diastole begins at the same region at which the contraction wave *originated*, and proceeds in the *same course* as did systole (Figures 8 and 9). Since the contraction wave reaches the tip of the appendix and the caudal border of the body nearly *simultaneously*, the auricle contracts symmetrically around the point of initiation of the wave. The body of the auricle is rather restricted in its movement, but usually contracts sufficiently to propel most, though not all, of its contents into the ventricle. On the other hand, the more mobile *auricular* appendix contracts more than the body during auricular systole. The exposed, *normally-beating* right auricle of the dog almost never empties completely during systole.

The vigor and *completeness of auricular systole* varies considerably from dog to dog. In rare instances, the right auricle appears to contract en masse so that the walls fade to a pale pink and the chamber is almost completely emptied of blood. Usually, however, auricular systole is less complete; the contraction waves are more or less lethargic, and their ability to propel blood into the ventricle is proportionately lessened. The reasons for this variation are not entirely clear. However, a few of the animals were in varying degrees of shock due to anesthesia, extensive surgical *procedure and* fluid loss. It may be that the auricular contractions in these animals were impaired by changes in blood volume, heart rate and *physiologic* surroundings of the heart, resulting from the operative procedures; such variations may not *exist in the intact* animal. Our experience with known shock states indicates that when the return flow of blood to the auricle is reduced, the vigor of auricular systole is correspondingly diminished

Because of possible anatomic and functional differences between species, it cannot *be* assumed that *observations on* the contraction of the auricles in the dog *apply also to man*. To establish the latter, evidence must be obtained directly by *visualization* of the human auricle or *indirectly* by other methods of investigation. *High* speed motion pictures were taken of the *human auricle on* four occasions during cardiac surgery. In each instance the human auricle appeared to behave like that of the dog.

Information derived from radiocardiographic *studies*[490, 491] seems to indicate that the human heart, like that of the dog, does not empty itself *completely during the systolic* phase of the cardiac cycle. When radioactive sodium (Na24) is injected intravenously into the human and its

human subjects during surgical procedures on the heart and lungs. This method of observation yielded direct and conclusive evidence regarding many features of the heart beat which hitherto had been subjects of speculation.

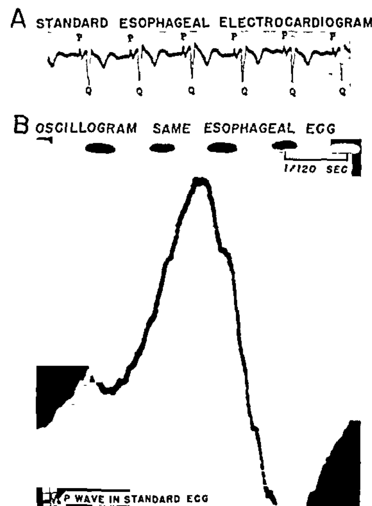


Figure 7. (A) Unipolar, esophageal lead from auricular level, normal male Standard electrocardiogram

(B) Same subject Auricular complex recorded and magnified by means of oscillograph. Camera speed increased from 25 mm per second to 381 mm per second Amplitude increased 32 times. Insert at lower left hand corner of B shows the same P wave obtained with standard electrocardiogram equipment

Observation 1: Site of Origin of the Normal Contraction Wave

As stated above, in 1907 Keith and Flack [303] suggested that the cardiac pacemaker might be located in the mass of specialized tissue found in the wall of the auricle near the superior vena cava. The suggestion was based upon the relatively large size of this mass and its intimate association with the many nerve fibers supplying the heart. However, up to the present time, no general agreement has been reached as to the *exact* site of origin of the cardiac impulse.

MacWilliam in 1887,[422] utilizing the known effects of temperature changes on the functional activities of tissues, found that the "application of slight heat locally to the terminal part of the vena cava superior gives a marked acceleration in the rhythm of the whole heart." Conversely in 1910 Flack [196] reported that a localized cooling agent slowed the rate of the heart only if it was applied to the region of the sino-auricular node. Gantner and Zahn in 1911 [214] likewise found that cooling of the sinus node slowed the rate of the heart beat; in addition, they noted that the farther the point of cooling was removed from the node, the less marked was its effect.

Lewis [357, 382, 385] and others,[50, 54, 55, 56, 99, 109, 171, 413, 414, 459, 679] investigated this problem by a variety of methods, including destruction of the sino-auricular node and determination of the *point of primary negativity in the auricle.* Lewis [385] found "abundant and conclusive evidence that the excitation wave begins in or near the head of the sino-auricular node." Eyster and Meek [177, 178, 179, 180, 182, 183, 184] confirmed many of Lewis' observations. On the other hand, Rijlant and Geraudel, according to Wiggers,[648] held the opinion that the normal beat actually arose from a restricted locus (1 millimeter in diameter) superior to the sino-auricular node. Wiggers [648] further stated that "while we may continue to regard the region of the sino-auricular node as the normal pacemaker, it is necessary to broaden our conception to include the possible origin of the impulse in adjacent tissue." To add further confusion, Glomset and Glomset [231] and others were unable to obtain any histologic evidence of the existence of a separate specilized sino-auricular node. Nonidez,[469] however, found histologic evidence of a node in hearts of puppies and in the monkey.

From electrocardiograms taken directly from the region of the sino-auricular node, the site of origin of the cardiac impulse cannot be determined. Deflections from the node, and those from points adjacent to it, resemble each other *too closely. Similarly indeterminate* are the

relatively crude cooling experiments.

By direct visualization of the dog's right auricle on high speed cinematographs, the *exact site* on the auricular surface of initiation of the contraction wave has been clearly seen for the first time. Motion pictures in 25 animals consistently showed that the *contraction wave starts in a region on the sulcus terminalis (corresponding to the taenia terminalis) at a distance approximately 1 centimeter to the right of the junction of the right auricular appendix with the superior vena cava.* This locus is nearly equidistant from the caudal and cephalic extremities of the auricle (Figure 8)

If the anatomy of the sino-auricular node in the dog corresponds to that usually described for man, the locus of initiation of the auricular contraction wave in the dog is about *halfway* between the head and tail of the node, perhaps somewhat closer to the tail. Contrary to the view now held by most workers, the wave does not originate *in* the head, as revealed by the films, the location of the head is approximately 1 centimeter to the left of the region in which the contraction wave is seen to arise.

Observation 2. Normal Contraction of the Right Auricle

In colored motion pictures taken at 1000 or 2000 frames per second, the first manifestation of auricular systole observed on the screen is a wrinkling of the body of the right auricle near the *tail* of the sino-auricular node (site of initiation of contraction wave). From this locus a wave of contraction spreads rapidly over the auricle in an ever-widening half-circle, completely engulfs that chamber, and leaves it contracted in all its dimensions. When the wave reaches the periphery of the auricle, maximum systole is attained. As the contraction subsides, relaxation into diastole begins at the same region at which the contraction wave originated, and proceeds in the same course as did systole (Figures 8 and 9). Since the contraction wave reaches the tip of the appendix and the caudal border of the body nearly simultaneously, the auricle contracts symmetrically around the point of initiation of the wave. The body of the auricle is rather restricted in its movement, but usually contracts sufficiently to propel most, though not all, of its contents into the ventricle. On the other hand, the more mobile auricular appendix contracts more than the body during auricular systole. The exposed, normally-beating right auricle of the dog almost never empties completely during systole.

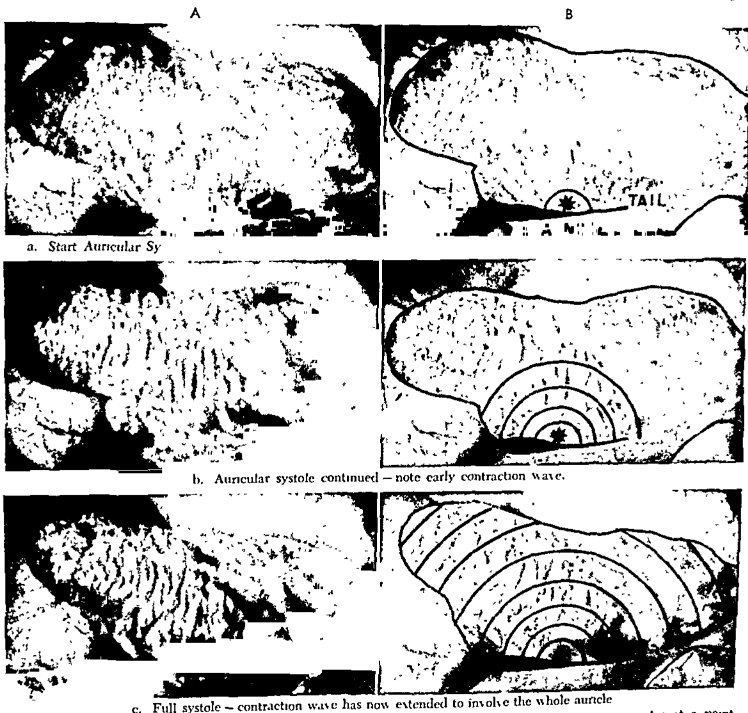
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course through the heart graphically recorded with an ink-writing Geiger-Muller counter, *the* inflow curve for the right heart is represented by a sharp upward deflection, the R wave; this *would indicate that the radiosodium* enters the right chambers rapidly. If emptying were complete with each systole, the subsequent downward deflection should be equally steep. In all instances, however, the outflow curve of the right heart is much more gradual (more horizontally inclined) than the inflow curves; when *plotted on semilogarithmic paper, it generally* inscribes a straight line (Figure 10). The conclusion thus appears justified that the right chambers of the human heart do not empty completely with each systole. It is more difficult to draw conclusions regarding the left heart from the configuration of the L wave in the radiocardiogram.

The inflow and outflow tracts of the right



a. *Start Auricular Sy*

b. Auricular systole continued – note early contraction wave.

c. Full systole – contraction wave has now extended to involve the whole auricle

Figure 8. (A) Enlarged photographs of right auricle showing progressive phases of auricular contraction

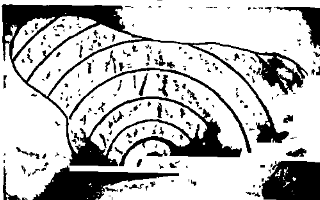
(B) Same as A with superimposed diagrams showing outlines of auricle during each phase, (a) point of initiation of contraction wave, and (b) and (c) successive phases of systole Note that contraction wave originates at a point equidistant *from* cephalic and caudal extremities of auricle. From this point the contraction wave spreads in an ever-widening half-circle and engulfs the entire auricle

ventricle can be easily seen in the motion pictures. As auricular systole reaches the halfway point in its course, a bulge forms in the wall of the right ventricle along the entire length of its attachment at the auriculo-ventricular groove. As auricular systole is completed, the bulge travels rapidly toward the cardiac apex and outlines the inflow tract of the right ventricle. As the blood reaches the apex of the right ventricle, ventricular systole begins; the blood then traverses the outflow tract of the ventricle into the pulmonary artery. In its course from the inflow to the outflow tract, and into the pulmonary artery, the blood stream actually changes direction approximately 300 to 325 degrees. The clarity with which the inflow tract can be seen in the motion pictures depends upon the vigor of auricular systole.

A



B



a Auricle in full *systole*.





b Mid diastole — note that the



c. *Complete* diastole

Figure 9 Continuation of events shown in Figure 8 As contraction wave subsides, relaxation into diastole begins at same point at which contraction wave originated and proceeds in the same manner as did systole

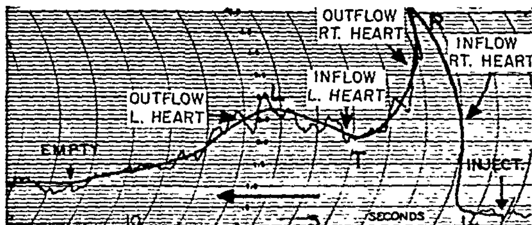


Figure 10. Radiocardiogram of normal human heart. Tracing reads from right to left. R wave represents inflow to (ascending limb) and outflow from (descending limb) right heart. L wave represents inflow to and outflow from (filling and emptying of) left heart. T represents transition phase when most of the blood is in pulmonary circulation. At point marked "empty" the tracing reaches a level plateau which represents exit of all radioactively labelled blood from heart. Note that inflow is more rapid than outflow in right heart

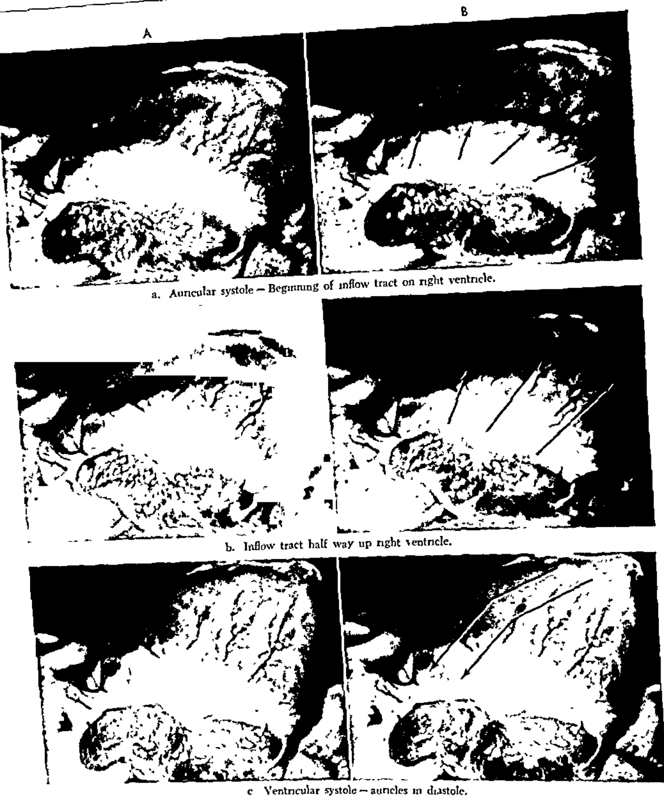
When systole is vigorous, the inflow and outflow tracts become prominent (Figure 11). No such inflow tract can be seen in the left ventricle, probably for two reasons: (1) the relative weakness of contraction of the left auricle; and (2) the greater thickness of the wall of the left ventricle.

On motion pictures taken at 2000 frames per second and projected at 16 frames per second, the actual speed of the contraction wave is reduced approximately 125 times. The speed with which the contraction wave travels in the motion picture may be measured by means of a stop-watch; by correcting for camera and projection speeds, the actual length of time taken for the wave to travel between any two points may be accurately calculated. Using this method, the contraction wave is found to travel from its site of initiation to the tip of the right auricular appendix in almost 0.01 second. The distance between the tip of the appendix and the origin of the impulse is approximately 3 centimeters. Hence, the speed of the normal auricular contraction wave is about 300 centimeters per second. On motion pictures taken at 2000 frames per second and projected at 16 frames per second, the average length of time occupied by auricular systole is 15 seconds and that consumed by auricular diastole is 40 seconds. After corrections for camera and projection speeds, the actual duration of these two phases of the cardiac cycle is found to equal 0.13 second for systole and 0.33 second for diastole. The time occupied by systole as calculated by this method closely approximates the figure of 0.11 second given by Wiggers.[640, 641, 643] The duration of the diastolic portion of the auricular cycle varies inversely and that of the systolic phase directly with the auricular rate. Thus, in intact animals with slower heart rates, diastole is proportionately longer and systole proportionately shorter than the figure given above.

Observation 3: Normal Contraction of the Left Auricle

Largely because of the difficulty in exposing the left auricle for experimental study, reported observations on this chamber are few. With the technique employed in this laboratory (see Appendix), the entire left auricular appendix, a major portion of the body of the left auricle, and several pulmonary veins were exposed and studied cinematographically.

As observed in the motion pictures, left auricular systole may be divided into those events which occur in the appendix and those which take place in the body. In the majority of dogs, movements rarely occur in the body of the left auricle during systole or diastole. In only a few cinematographs are occasional ripples of



a. Auricular systole — Beginning of inflow tract on right ventricle.

b. Inflow tract half way up right ventricle.

c Ventricular systole — auricles in diastole.

Figure 11 Right auricle in systole propelling blood into right ventricle which is in diastole. Diagrams on the right outline the progressive movement of blood through inflow and outflow tracts of right ventricle during phase of ventricular filling.

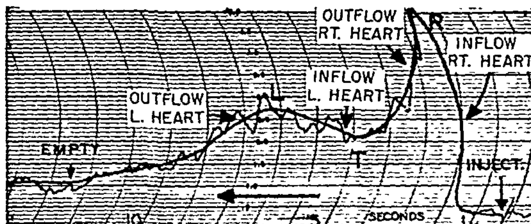


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dix relaxes somewhat more slowly than it had contracted in systole, and becomes distended with blood (Figure 12).

When both auricular appendices are observed in the same cinematograph they appear to contract simultaneously. As in the right auricle, there is considerable variation from animal to animal in the force and completeness of left auricular systole. In a few hearts, the appendix contracts to such an extent that it pales to a light pink color. In the remaining hearts, contractions are variably less intense; in some instances systolic discharge is only about 50 per cent of the content of the appendix. Again, as in the right chamber, increased heart rate, varying degrees of shock, and difference in fluid loss may be wholly or partly responsible for these variations.

Electrocardiographic Correlation: Principle of Dissociation: By means of direct auricular lead electrocardiograms, we have repeatedly demonstrated that electrical activity during auricular systole may be identical in the body of each auricle. Since the body of the left auricle is almost motionless and that of the right exhibits comparatively vigorous contractions, it appears that *electrical activity may exist with little or no apparent muscular movement.* During our experiments, however, we have never observed muscular movement without associated electrical activity. These observations have been made by simultaneous motion pictures and electrocardiograms of both auricles under a wide variety of conditions, both normal and abnormal, in at least 25 experiments, and demonstrate the principle that electrical activity need not be accompanied by apparent muscular movement

OBSERVATION 4: THE FUNCTION OF THE INTERAURICULAR SEPTUM

The anatomic unity of the interauricular septum with the auricles proper has been described earlier in this chapter. The following experiments were designed to study the conductile and contractile properties of the septum in relation to total auricular function.

Electrical Activity of the Interauricular Septum: The heart was exposed in the manner described in the Appendix. Through an incision in the right auricular appendix, a nonpolarizable electrode was placed against the septum. A purse-string suture was drawn around the electrode to prevent bleeding. It was determined by palpation that the electrode was in direct contact with the septum. Electro-

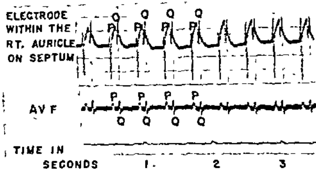


Figure 13. Direct intra-auricular lead electrocardiogram. Tip of electrode is on interauricular septum. Lead AVF was recorded simultaneously. Note that electric impulse is conducted through interauricular septum.

cardiograms taken from this electrode *revealed auricular deflections essentially identical with those obtained elsewhere on the walls of the auricles.* Since these deflections were of the type which can be obtained only when the cardiac impulse passes directly beneath the recording electrode (Chapters II, VI and VII), it is apparent that the interauricular septum conducts the cardiac impulse in the same manner as the remainder of the auricles (Figure 13).

As frequently noted in the text, there is often dissociation between electrical and mechanical events occurring in the auricles; normal electrocardiographic complexes may be recorded from the auricles without corresponding auricular contractions. Hence, although the preceding experiment established that the interauricular septum conducts the cardiac impulse, it did not prove that the septum participates in auricular contraction. To establish the latter fact, actual visualization of the septum was necessary. This was done in the following cinematographically recorded experiment performed in two dogs.

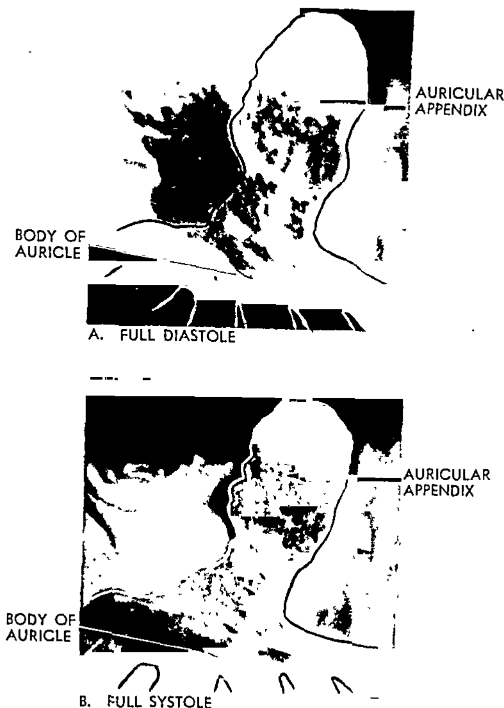


Figure 12. Enlarged photograph of left auricle taken from high speed cinematograph (A) *Full diastole* Note situation of body which is bound down by pulmonary veins and long auricular appendix. (B) Full systole of left auricle Note that left auricular appendix narrows and contracts down somewhat but body does not move.

contraction seen; these are at best only abortive attempts at an organized systole. Thus, the body of the left auricle in the dog appears to serve mainly *as a conduit and not as a contractile organ; the entire effective motion of the left auricle is that of the appendix.*

At the onset of auricular systole, the first movement is a wrinkling at the base of the appendix. This wrinkling spreads rapidly and reaches all points on the periphery simultaneously. As the contraction waves reach the periphery, the appendix is rapidly drawn in and down toward its base. The surface of the appendix now has been thrown into a myriad of folds or wrinkles. The body *is undisturbed,* remaining as it was in diastole. After a pause, diastole begins in the area on the auricle where the contraction wave was initiated; the appen-



a. Diastole.



b First systolic contraction



c Interruption of systole by relaxation.



d Second systolic contraction

Figure 15 Enlarged photographs of left auricle from cinematographs, showing "double contraction" of auricle in systole (a) Auricle is in diastole, (b) contraction has just started, (c) systole is interrupted during brief period of relaxation, and (d) completion of contraction.

locking mechanism highly efficient, since the taenia contracts at the instant auricular systole begins. On the other hand, if the contraction wave originates at some abnormal point on the auricle, a reflux of blood into the venae cavae occurs during the period of systole preceding activation of the taenia.

(3) The speed of propagation of the contraction wave in normal sinus rhythm is greater than in any of the auricular arrhythmias; as a consequence the wave invades all portions of the chamber at virtually the same time and results in efficient auricular contraction and emptying. When the contraction wave traverses the auricle more slowly, as it does in the higher-rate auricular arrhythmias, the auricle tends to contract unsymmetrically and blood is diverted to dilated portions of the chamber. While a significant amount of blood finds its way through the auriculo-ventricular orifice, a large proportion of auricular energy is wastefully dissipated.

The foregoing description emphasizes the strategic value of the normal location of the pacemaker within the sino-auricular node, namely, approximately halfway between the head and tail, probably somewhat closer to the latter. The auricular contraction would be less efficient if it arose in the head proper, which is relatively less central with reference to the caudal and cephalic extremities of the auricle. To demonstrate this point, the following experiment was performed:

Efficiency of Beat Arising from Head of Sino-Auricular Node: A fine, copper-wire, stimulating electrode was inserted into the head of the sino-auricular node at the junction of the appendix and the superior vena cava; single induction shocks from a pulse generator were sent into the auricle at a rate of 75 per minute. Stimuli at this rate elicit occasional premature systoles originating at the point of stimulation. Cinematographs taken at 2000 frames per second during the period of stimulation unequivocally showed that the contraction wave starting at the head of the sino-auricular node is much less efficient than the wave starting at the normal site. Because its origin is relatively less central and near the right auricular appendix, the abnormal contraction wave envelops the appendix before the body; when the wave reaches the caudal end of the auricle, the body contracts and the appendix dilates. This causes a fruitless shuttling of blood from one portion of the auricle to another with much waste of auricular energy. On the other hand, when the wave starts at the normal focus near the tail of the sinus node, the auricle contracts symmetrically, literally funneling the auricular contents through the tricuspid valve into the ventricle.

The motion pictures disclose little concerning the efficiency of the left auricle during the various types of auricular systole. The appendix seems to contract in the same manner during normal sinus rhythm, auricular tachycardia, and auricular flutter. Since the body exhibits little or no motion, it is probable that the over-all efficiency of the left auricle is less impaired than that of the right during these auricular arrhythmias.

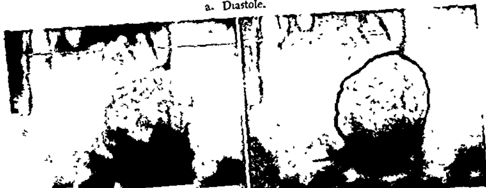
The observations on the efficiency of the normally-originating auricular contractions are perhaps more of physiologic than of clinical interest. In the well-functioning human heart, the onset of an arrhythmia such as an auricular tachycardia may have no noticeable effect on myocardial efficiency. In patients with incipient decompensations, however, the onset of the arrhythmia may precipitate overt failure. More detailed discussions of the efficiency of contraction waves in relation to specific auricular disturbances are found in the appropriate chapters.

Observation 8: Double Auricular Contractions

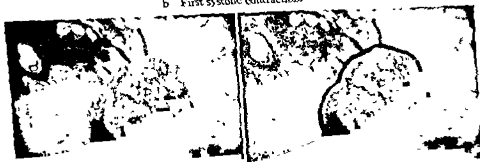
As described earlier, the usual normal auricular systole is a smooth, continuous contraction. However, cinematographic study (most revealing in this instance at 1000 frames per second) has disclosed that in some normal animals, auricular systole is in effect a double contraction consisting of two discrete partial movements (Figure 15). Two types of double auricular contractions have been observed. In each, au-



a. Diastole.



b First systolic contraction.



c Interruption of systole by relaxation.



d. Second systolic contraction.

Figure 15 Enlarged photographs of left auricle from cinematographs, showing "double contraction" of auricle in systole (a) Auricle is in diastole, (b) contraction has just started, (c) systole is interrupted during brief period of relaxation, and (d) completion of contraction.

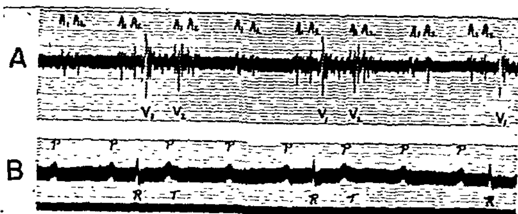


Figure 16. This figure shows a phonocardiogram and simultaneously recorded electrocardiogram from a patient with heart block. The auricular sound waves recorded during the auricular contraction show two distinct sounds, A_1 and A_2. It is probable that double auricular contractions are responsible for this phenomenon. (From Lewis: *Mechanism and Graphic Registration of the Heart Beat*, courtesy Shaw and Sons, Ltd, London.)

ricular systole is initiated in the usual manner. Thereafter, events proceed as follows:

(1) After a partial contraction the auricle *suddenly pauses*, but *almost in the same instant* resumes its activity and completes systole with full force and vigor.

(2) After a partial contraction the auricle suddenly relaxes into nearly full diastole, but immediately thereafter systole is resumed and completed in a smooth and regular manner.

In one cinematograph in which both auricles were photographed, *there was seen a double* contraction of the left auricular appendix but not of the right auricle (either body or appendix). The *single contraction of the right auricle* took place during the pause between the two phases of the double contraction of the left; it occurred *late in the interval, just before the* second contraction began. Since the existence of double auricular contractions was discovered, this phenomenon was recognized on one occasion in the left auricle of a dog as the exposed heart was observed with the unaided eye. In view of this experience, it appears possible that *cardiovascular surgery* will afford opportunities for similar observations in man. The phenomenon might also be studied clinically by electrokymography and by cardiac catheterization.

The mechanism of the double contraction is not clear. In the absence of concomitant electrocardiographic data, it cannot be stated whether or not the double mechanical systole is correlated with a double auricular electrical impulse. If double auricular systoles are found *to be accompanied by a single electrical impulse (which is probable since we consistently found* only one impulse for each double contraction), present views concerning the all-or-none law of cardiac muscle will require modification. Any speculation in this regard must await further study.

Two sounds during auricular systole have *been recorded in patients with heart block (Figure 16); it is possible that a double au*ricular contraction is responsible for this occurrence, Groedel[251] has recorded simultaneous jugular vein tracings and electrocardiograms during flutter (*Figure 17*). The records clearly show two distinct auricular waves in the jugu*lar vein tracing* for each auricular electrical wave. The explanation in this instance must be that two auricular contractions occur in each auricular systole. We have observed an apparently similar phenomenon in cinematographs of flutter in dogs. *Thus, double auricular contractions* probably occur in the human. Their clinical significance is yet to be elucidated.

THE ROLE OF THE AURICLES IN THE OVER-ALL CARDIAC FUNCTION

In many patients with auricular fibrillation, adequate cardiac compensation may be maintained for years if the ventricular rate remains within normal limits (spontaneously or with



Figure 17. Simultaneous jugular vein tracing (B.J.) and electrocardiogram (C.B.) during auricular flutter Two distinct auricular waves (a,a) are seen in the jugular vein tracing which correspond to each auricular deflection in the electrocardiogram The only possible explanations of this phenomenon are. (1) the auricles were contracting asynchronously, or (2) double auricular contractions were occurring. (From Groedel: *Venous Pulse*, courtesy Brooklyn Medical Press, New York)

digitalis). Because of this fact, and because the fibrillating auricles are believed by some investigators to undergo no significant contractions, many physiologists and clinicians consider the work of the normal auricles relatively unimportant in the over-all cardiac function. Such a conclusion is not justified without more specific investigation.

By the method of high-speed cinematography, the function of the auricles in the total cardiodynamics may be clearly determined. The amplitude and fullness of auricular systole, the degree of diastolic filling, and the completeness of auricular emptying can be well visualized on the films. The force and effectiveness of auricular contraction may be further estimated by noting the degree of ballooning of the inflow tract in the right ventricle (Figure 11). Thus, an accurate appraisal of the output of blood from the auricles under varying conditions can be obtained.

The contractile properties of the auricles were studied under conditions of (1) diminished work; and (2) increased work leading to heart failure.

Auricular Contraction Under Conditions of Diminished Work: In many experiments in which cinematographs were made during normal auricular systole, varying degrees of shock and sinus tachycardia were present. Under such circumstances the venous return was decreased and the auricles, functioning under conditions of diminished work, were seen on the films to contract poorly. Nevertheless, the venous return to the ventricles was maintained adequately, apparently with little or no help from the auricles. From such evidence one may form the impression that normal auricular systole is of relatively minor importance in the over-all cardiodynamics.

Auricular Contraction Under Conditions of Increased Work: Control motion pictures were taken of the exposed beating hearts in five dogs. Infusion of warmed physiologic saline solution was then started into the femoral vein at a rate of 100 to 150 cubic centimeters per minute (a method utilized by Tinsley Harrison[201] in the production of heart failure). On subsequent photographs, taken at three to five minute intervals, the following course of events was observed:

At the outset of the experiment, the right auricle and the left auricular appendix contract normally and participate in transporting the normal venous return to the ventricles. As described earlier, the body of the left auricle is relatively non-contractile and serves mainly as a conduit As the infusion progresses and the venous return increases, the right auricle and the left auricular *appendix* respond with vigorous and full [illegible]

prop [illegible]

volu. [illegible] ventricles. As the diastolic blood volume of the auricles continues to increase, auricular contractions become more and more vigorous, and for a time effectively compensate for the progressively enlarging venous return. During this period the stroke volume is greatly increased; the maximum diastolic size of the auricles is *much* greater and the systolic size much smaller than normal. It is strikingly apparent that under the stress of greatly increased



Figure 16 This figure shows a phonocardiogram and simultaneously recorded electrocardiogram from a patient with heart block. The auricular sound waves recorded during the auricular contraction show two distinct sounds, A_1 and A_2. It is probable that double auricular contractions are responsible for this phenomenon. (From Lewis: *Mechanism and Graphic Registration of the Heart Beat,* courtesy Shaw and Sons, Ltd , London)

ricular systole is initiated in the usual manner. Thereafter, events proceed as follows:

(1) After a partial contraction the auricle suddenly pauses, but almost in the same instant resumes its activity and completes systole with full force and vigor.

(2) After a partial contraction the auricle suddenly relaxes into nearly full diastole, but immediately thereafter systole is resumed and completed in a smooth and regular manner.

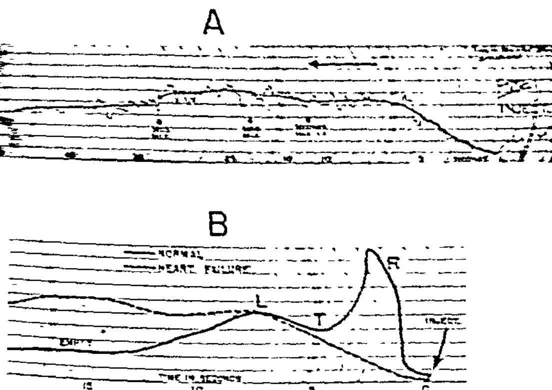
In one cinematograph in which both auricles were photographed, there was seen a double contraction of the left auricular appendix but not of the right auricle (either body or appendix). The single contraction of the right auricle took place during the pause between the two phases of the double contraction of the left; it occurred late in the interval, just before the second contraction began. Since the existence of double auricular contractions was discovered, this phenomenon was recognized on one occasion in the left auricle of a dog as the exposed heart was observed with the unaided eye. In view of this experience, it appears possible that cardiovascular surgery will afford opportunities for similar observations in man The phenomenon might also be studied clinically by electrokymography and by cardiac catheterization.

The mechanism of the double contraction is not clear. In the absence of concomitant electrocardiographic data, it cannot be stated whether or not the double mechanical systole is correlated with a double auricular electrical impulse. If double auricular systoles are found to be accompanied by a single electrical impulse (which is probable since we consistently found only one impulse for each double contraction), present views concerning the all-or-none law of cardiac muscle will require modification. Any speculation in this regard must await further study.

Two sounds during auricular systole have been recorded in patients with heart block (Figure 16); it is possible that a double auricular contraction is responsible for this occurrence, Groedel[251] has recorded simultaneous jugular vein tracings and electrocardiograms during flutter (Figure 17). The records clearly show two distinct auricular waves in the jugular vein tracing for each auricular electrical wave. The explanation in this instance must be that two auricular contractions occur in each auricular systole. We have observed an apparently similar phenomenon in cinematographs of flutter in dogs Thus, double auricular contractions probably occur in the human Their clinical significance is yet to be elucidated.

THE ROLE OF THE AURICLES IN THE OVER-ALL CARDIAC FUNCTION

In many patients with auricular fibrillation, adequate cardiac compensation may be maintained for years if the ventricular rate remains within normal limits (spontaneously or with



[illegible] A. Radiocardiogram of patient with congestive heart failure, arteriosclerotic heart disease and old myocardial infarction. Note that curve rises gradually due to [illegible] of heart and that R and L waves merge; curve [illegible] due to slow emptying of the heart. [illegible] have been removed from tracing, at the [illegible] to permit reproduction.

B. Graphic visualization of abnormal radiocardiogram [illegible] superimposed on normal radiocardiogram. The solid line is the normal and the broken line the abnormal record. In the normal record the curves of right R and left heart filling L and the transition point T are clearly demarcated. In the record from the patient with congestive heart failure the curve rises slowly, the right heart fills poorly, no definite transition point is seen, and emptying, once filling has been achieved, is prolonged. The normal heart has completely emptied within 15 seconds.

Figure 13. Enlargements from high-speed cinematographs showing effect of increased work on contraction of right auricle. Diastole is depicted on left, systole on right. The top photographs show normal cycle: The auricle in full diastole is relatively small and pale in color; during systole the appendix is moderately pulled in and contracted downward with body of auricle which is incompletely contracted. The center photographs, following infusion of warm physiologic saline solution at rate of 100 to 150 cubic centimeters per minute. During diastole the auricle is larger than in normal cycle; in systole the auricle is smaller than in normal cycle. Note tremendous dilation of right ventricle at completion of vigorous auricular systole. This series of photographs illustrates that the amount of contraction is proportional to the degree of diastolic filling (Starling's Law) and demonstrates that under conditions of stress the auricles are capable of considerable work.

The *lower photographs*: Heart in failure due to continued infusion. Note that diastolic volume is considerably greater than in the normal cycle, and during systole the auricle is able to contract little if at all. The right ventricle is contracted but to a lesser degree than in the normal cycle; the contraction of the left ventricle is evident.

venous return, the auricles respond with great force and vigor to maintain the integrity of the circulation, many times the normal auricular content is literally forced into the ventricles by the increased strength of auricular systole (Figures 13A and B.) The augmented auricular activity causes the right ventricle to balloon immediately following auricular systole. As the venous return continues to increase, a point is finally reached at which the intensity and completeness of auricular systole begin to diminish as the auricle continues to dilate; *soon all evidence of auricular contractions completely disappears* (Figure 13B.) Simultaneously recorded electrocardiograms show that electrical activity continues undisturbed.

The direct visual evidence that within certain physiologic limits, auricular contractions grow

DIASTOLE SYSTOLE



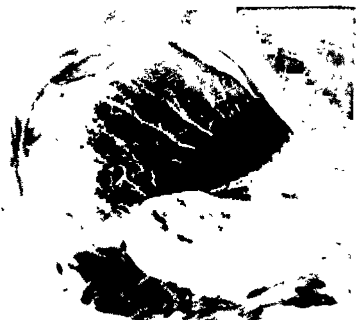


Normal cardiac cycle – Auricular diastole on left, systole on right.





Following infusion of saline solution – Note increased auricular volume in diastole and increased auricular systolic contraction





Infusion continued – Heart failure present – Note poor *auricular contraction*

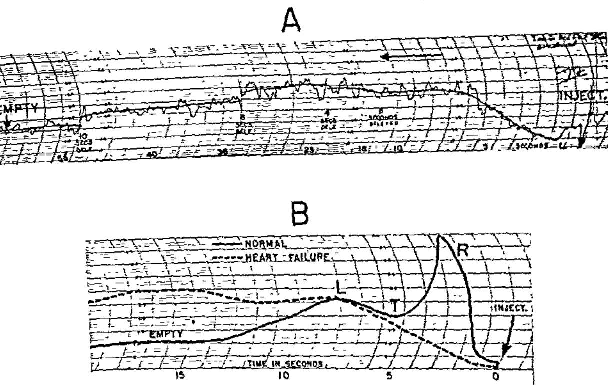


Figure 19 A. Radiocardiogram of patient with congestive heart failure, arteriosclerotic heart disease and old myocardial infarction. Note that curve rises gradually due to

segments

B Graphic visualization of abnormal radiocardiogram in A superimposed on normal radiocardiogram. The solid line is the normal and the broken line the abnormal record. In the normal record the curves of right R and left heart filling L and the transition point T are clearly demarcated. In the

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venous return, the auricles respond with great force and vigor to maintain the integrity of *the circulation; many times* the normal auricular content is literally forced into the ventricles by the *increased strength* of auricular systole (Figures 18A and B). The augmented auricular activity causes the right ventricle to balloon immediately following auricular systole. As the venous return continues to increase, a point is finally reached at which the intensity and completeness of auricular systole begin to diminish as the auricle continues to dilate, *soon all evidence of auricular contractions completely disappears* (Figure 18B). Simultaneously recorded electrocardiograms show that electrical activity continues undisturbed.

The direct visual evidence that within certain physiologic *limits*, auricular contractions grow

more vigorous and complete as the diastolic volume increases, and more feeble and incomplete as the diastolic volume decreases, confirms the applicability to the auricles of a well-known physiologic principle first defined by Starling (Starling's "law of the heart").

Clinical Correlations: From the above series of observations, it is evident that the stroke output of the auricles in the dog may vary within wide limits. When shock or tachycardia is present and the venous return small, the stroke output of the auricles diminishes and auricular contraction is relatively weak. When the venous return is larger and the diastolic auricular volume correspondingly increases, contractions become full and vigorous and the auricular output is greatly augmented. Eventually, progressively increasing diastolic volume may stretch the auricular wall to a point at which spontaneous compensation is no longer possible, and auricular failure results. That these experimentally demonstrated phenomena are applicable to man, as well as the dog, is indicated by both radiocardiographic and clinical evidence.

Clinicians have noted fluoroscopically that the amplitude of auricular contraction is decreased in shock states and in congestive heart failure, while in conditions associated with excitement the amplitude of auricular contraction is greatly increased. The existence of these phenomena, long known clinically, has now been confirmed by experimental production and direct cinematographic observation in the dog.

Since the dog's auricle normally fails to empty itself completely during systole, obviously emptying would be even more incomplete in auricular failure. As noted earlier, radiocardiographic study demonstrates that the normal human heart likewise does not empty itself completely. By the same method, it can be shown that the amount of blood remaining in the auricle is much greater in clinical congestive failure. In Figures 10 and 19B (radiocardiograph of normal human heart), the angle at R (end-point of right heart filling) is extremely acute; the angle at L (end-point of left heart filling) is relatively obtuse. Examination of the abnormal curve representing congestive failure, as shown in Figures 19A and B, discloses that no angle is present at R and angle L is flattened to almost 180 degrees. Thus, comparison of the curve from a patient in severe congestive failure with that of the normal demonstrates that the filling of the heart in congestive failure is gradual and slow with no definite end-point in either the right or left side of the heart and that the emptying is extremely prolonged. The analogy of these altered cardiodynamics in the human with those observed in the experimentally induced cardiac failure in the dog is apparent.

As first observed by Mackenzie,[116] the diastolic rumbling murmur of mitral stenosis may diminish or disappear when congestive failure is advanced. The murmur recurs with its previous intensity after congestive failure is alleviated. Since authorities agree that the murmur is due to vibrations created as the blood goes through the narrowed mitral valve, apparently less blood passes through the valve with less vigor during congestive failure. Therefore, it may be that in man, as in the dog, the auricle contracts less completely when congestive failure is present.

In the normal phonocardiogram a sound wave precedes the first heart sound. This sound wave is attributed to auricular systole, in the absence of cardiovascular abnormalities it is usually below the range of clinical audibility. This is not surprising in view of the comparative weakness of normal auricular systole In conditions associated with increased auricular activity, it is probable that the greater force and excursion of the auricular contractions increase the amplitude of this auricular sound wave and render it audible. Since diastolic gallop rhythms are associated with increased auricular activity, this may be the mechanism responsible in some cases for the third sound characteristic of such rhythms. This mechanism can be assumed to operate in clinical ventricular failure. As a result of ventricular failure, the auricles are called upon to perform increased amounts of work, the amplitude of auricular systole is augmented, and the third sound becomes audible.

The amount of work done by a hypertrophied auricle, such as commonly occurs in certain forms of heart disease in which the auricle has been under prolonged strain, is undoubtedly greater than that in acute experiments on dogs. This would explain the frequent occurrence of some types of gallop rhythm in patients with hypertrophied auricles.

The comparative rarity of gallop rhythm during auricular fibrillation, in which there is no organized electrical or mechanical activity, is also consistent with these observations

SUMMARY AND CONCLUSION

By means of high-speed cinematographs, the exact site of origin of the normal heartbeat in the dog has been visualized for the first time. It appears to lie in the taenia terminalis between the head and the tail of the sino-auricular node, somewhat nearer the tail, at a site approximately midway between the caudal and cephalic extremities of the auricle.

The dynamics of right and left auricular contractions, as observed in slow motion pictures, has been described The right auricle contracts symmetrically about the site of origin of the impulse, the left auricular appendix contracts symmetrically about its base. The auricles usually do not empty completely during sinus rhythm. The body of the left auricle in the dog is largely non-contractile and serves mainly as a conduit The interauricular septum is anatomically and functionally an integral part of both auricular chambers, like the remainder of the auricular musculature, it both conducts the cardiac impulse and undergoes contraction.

Auricular contractions arising at the normal site of origin are more efficient than those from other foci on the auricle, including the head of the sino-auricular node. The following three factors combine to make normal sinus rhythm the most efficient auricular mechanism. (1) the site of origin of the contraction wave near the tail of the sino-auricular node, midway between the cephalic and caudal ends of the right auricle, allows the wave to reach the extremities of the auricle nearly simultaneously, (2) by means of a venous-locking mechanism, the contraction of the taenia terminalis at the onset of auricular systole prevents reflux of blood into the venae cavae (this sphincter-like mechanism does not occur in the left auricle); and (3) due to the relatively rapid speed of propagation of the normal contraction wave all portions of the auricles enter systole nearly simultaneously. In the auricular arrhythmias, one or more of these three factors is lacking; as a result, the contraction waves are less efficient.

An interesting incidental finding of undetermined significance is the discovery that in some normal animals auricular systole consists of two distinct partial contractions.

The cinematographic technique of studying auricular contraction offers a direct visual method of determining the role of the auricle in the over-all cardiac function. Evidence obtained by this method appears to indicate that under normal conditions the exposed dog's auricles play a relatively unimportant part in the transportation of blood to the ventricles; under conditions of stress (increased venous return or ventricular failure) they assume a role of major importance in maintaining the integrity of the circulation by contracting with force and vigor. During periods of diminished work the auricles contract poorly. As the diastolic volume grows progressively larger, finally the auricles can no longer respond and complete failure ensues; motion pictures reveal that the auricles at this state are greatly dilated and no longer contract.

The electrocardiographic characteristics of normal sinus rhythm are briefly described. Although the body of the left auricle is almost motionless during normal systole while that of the right contracts vigorously, direct lead electrocardiograms indicate that electrical activity in the bodies of the auricles is identical. Similarly, when auricular failure results from the stress of increased venous return, electrical activity in the auricles is undiminished. These two observations demonstrate the principle of dissociation of mechanical and electrical activity. Mechanical activity in the auricles may be greatly diminished while normal electrical activity persists unchanged.

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disease with mitral stenosis, the disturbance may presage the appearance of auricular fibrillation[49] Multifocal premature beats may accompany severe myocardial disease but we have also seen such beats in normal hearts. An increase in the number of premature contractions following exercise suggests a damaged myocardium.[49] Auricular premature systoles have been reported to result from exercise in patients with angina pectoris,[492] in these instances the development of the premature contractions preceded the onset of anginal pain and could be prevented or delayed by quinidine. The prognostic significance of the arrhythmia appears to be related to the cardiac rate, in patients with heart rates over 110 per minute, the presence of auricular premature beats may indicate myocardial damage. Auricular premature contractions may be associated with auricular infarction.[121]

In nervous and sensitive subjects the symptoms may be extremely annoying and often become the basis for a cardiac neurosis. A wide variety of sensations may be experienced, such as. (1) palpitation caused by the first strong beat after the pause;[205] (2) cerebral symptoms following the sudden damming of blood in the jugular veins if the auricle contracts during ventricular systole; and (3) a minor fluttering sensation which may occur during an interpolated premature systole.[205, 665] In many individuals these symptoms gradually disappear although the arrhythmia continues. In rare instances premature systoles occur with such frequency as to interfere with the cardiac output and cause giddiness

The clinical diagnosis can be made more accurately by direct cardiac auscultation than by palpation of the peripheral pulse, in the peripheral artery the premature beat may be delayed, weak or imperceptible.[205, 343, 638] By either method, when auricular premature systoles are numerous they are sometimes difficult to distinguish from auricular fibrillation. A differential diagnosis is easily made by increasing the heart rate. As a rule, simple exercise will cause auricular premature systoles to disappear but will increase the irregularity of fibrillation.

Individual premature beats may be too weak to open the semi-lunar valves. Under such circumstances two phenomena may occur: (1) Such beats fail to reach the peripheral pulse, as a result of which fewer beats are palpable at the wrist than are audible at the cardiac apex ("pulse deficit") and (2) at the apex, only the first heart sound might be heard. Levine and Harvey[346] have pointed out that in rare instances auricular premature systoles may occur without accompanying heart sound.

The difference in length of the compensatory pulse sometimes enables a keen observer to make a bedside differential diagnosis between auricular and ventricular premature systoles. The compensatory pause after a auricular premature systole is usually short; in ventricular premature systole the time interval from the preceding sinus beat through the compensatory pause is exactly equal to the time occupied by two normal beats.

Electrocardiographic diagnosis is, of course, the most conclusive. Nevertheless, the major portion of our fundamental knowledge was obtained through studies of the venous and arterial pulse by means of the polygraph and sphygmograph long before the electrocardiograph had come into general use.[412, 629]

Elimination of aggravating factors together with reassurance are usually the only measures required for successful therapy. The use of various pharmacologic agents in the treatment of this arrhythmia is discussed in Chapter XVII.

Because of the innocuous character of the auricular premature systole, few studies have been devoted to the elucidation of its exact mechanism. In this monograph, however, auricular premature systole is given major emphasis, for a clear understanding of its nature is fundamental to the comprehension of the more complicated auricular arrhythmias.

EXPERIMENTAL METHODS

Experimentally produced auricular premature systoles in over 40 dogs were studied by means of high-speed cinematography, electro-

CHAPTER II

Auricular Premature Systole

AURICULAR premature systole — the simplest auricular arrhythmia — was among the first of the cardiac disorders investigated by means of the new techniques and apparatus developed in the late nineteenth century. Brief references to irregularities in the cardiac rhythm are found in the earliest medical writings.[273] Clinical studies of premature contractions based on the improved methods were carried out by Knoll in 1872[312] and Rosenstein in 1877, 1879. During this period the primary discoveries of Bowditch in 1871[59] on the all-or-none law; of Marey in 1877;[432] and of Cushny and Matthews in 1897[127] on the refractory phase of cardiac activity and of Engelman in 1894, 1895[164, 165] on the physiology of the pulsating heart, enabled investigators to establish fundamental facts relating to the auricular premature systole. Sir James Mackenzie in 1894[411, 414] differentiated auricular and ventricular premature contractions, Wenckebach in 1898[627, 628] and, independently, Cushny in 1899[122] discovered that the clinically observed premature contractions of the heart are similar to those obtained experimentally in the physiology laboratory. Perhaps the greatest clinical contribution of the period was that of Mackenzie,[412] who first demonstrated that auricular premature systole was of itself harmless and bore none of the grave prognostic implications which most clinicians had attributed to it.

Many other workers in the late nineteenth century, including Henschen, Langendorff and Hering, made valuable studies in this field.[629] In view of the limited facilities at their disposal the observations were remarkably accurate; their basic conclusions are consistent with results obtained by use of the most modern investigative methods.

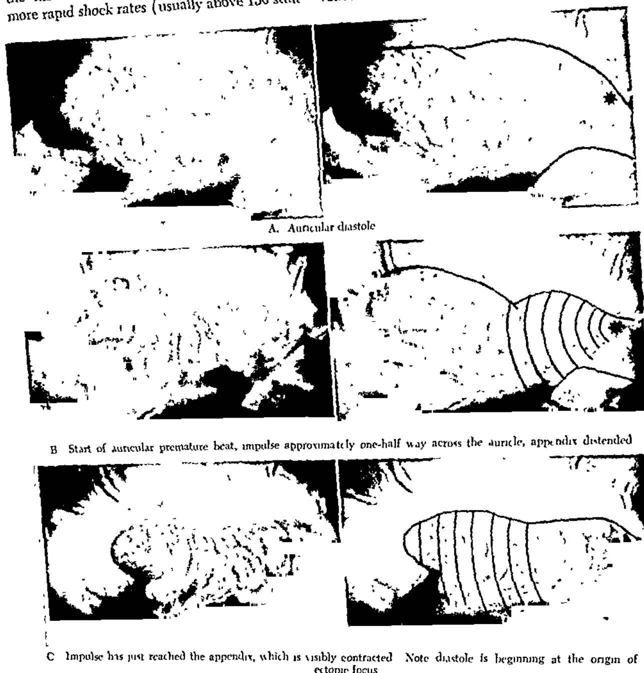
CLINICAL CONSIDERATIONS

Auricular premature systoles occur somewhat more frequently in males than in females. The incidence is highest in the aged, lowest in infants; no age is exempt. In the majority of patients the heart is structurally normal;[89, 205, 345, 441, 638] in the presence of heart disease, however, the incidence of auricular premature systoles is greater than in a comparable group of normal persons. Approximately one-half of all patients with this disturbance have no knowledge of its existence.

The etiology is unknown. In susceptible individuals various extraneous aggravating factors, such as tobacco and alcohol, may initiate the arrhythmia. Forced breathing may elicit premature beats.[559] Instances have been noted in hyperthyroid[307] and in myxedematous patients.[602] Surgical procedures are among the common inducing factors; the disturbance has been precipitated by intubation and anesthesia,[496] intrathoracic operations,[81] pericardiectomy[587] and cardiac catheterization[240, 268, 308, 494] Auricular premature systoles have occurred as a result of digitalis poisoning.[83] When associated with infection, such beats apparently indicate cardiac involvement but not necessarily irreversible damage.[49]

Auricular premature systole in normal hearts usually is inconsequential. In general, the presence of this arrhythmia in individuals with cardiac or other disease does not add to the gravity of the prognosis.[174] When associated with certain heart conditions, especially rheumatic heart

ber of effective electrical stimuli relatively fewer; the basic cardiac rhythm remained under the influence of the sino-auricular node. At more rapid shock rates (usually above 150 stimuli per minute) the artificially stimulated ectopic focus generally became the pacemaker and auricular paroxysmal tachycardia supervened. The critical level at which the ectopic



A. Auricular diastole

B Start of auricular premature beat, impulse approximately one-half way across the auricle, appendix distended

C Impulse has just reached the appendix, which is visibly contracted Note diastole is beginning at the origin of the ectopic focus

Figure 21 Cinematographic appearance of the auricular premature systole arising from an ectopic focus (induced by electrical stimulation) at extreme caudal end *of right* auricle just ventral to the inferior vena cava In column to the left are stills taken from cinematographs In column to the right are same stills with diagrammatic illustrations superimposed From its point of origin the premature systole spreads in a broad band involving entire width of auricle Diastole likewise starts at the ectopic focus and travels in the same direction as systole

A shows auricle in complete diastole. B shows con-
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cardiography and cathode-ray oscillography. In most instances the more rapid rate of arrhythmias were produced in the same animals following completion of the observations on auricular premature systoles. Three methods were utilized in the production of premature beats:

(1) Mechanical irritation of the auricle by stroking with a wooden applicator.

(2) Local application of 0.05 per cent aconitine in benzene to a small area on the wall of the auricle.

(3) Electrical stimulation through a fine copper-wire electrode sutured to the auricle at a chosen site.

The auricle was stimulated by single induction shocks administered at a determined rate by means of a faradic stimulator. Shocks at rates under 100 per minute commonly produced occasional and scattered auricular premature beats. Only a few of the stimuli found excitable muscle to produce premature systoles; the majority fell during refractory periods and were ineffective. During rapid sinus rhythms, the non-refractory (excitable) periods were relatively shorter and consequently the num-

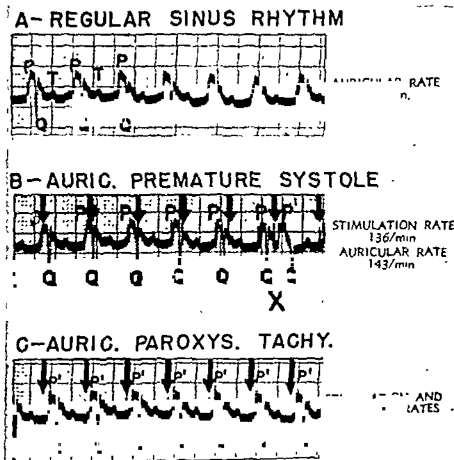


Figure 20 Three direct auricular electrocardiograms illustrating the effect on the heart rate produced by changes in rate of electrical stimulation

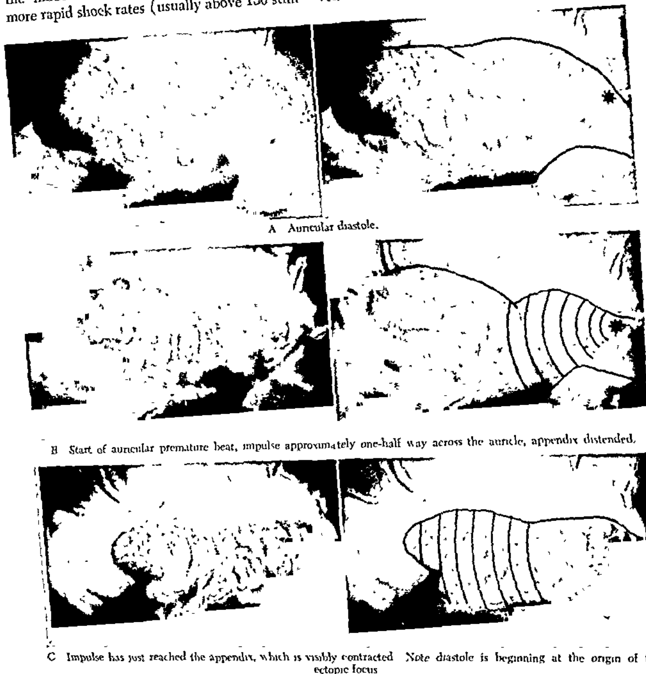
A Normal sinus rhythm, auricular rate 130 beats per minute

B Auricular premature systole Auricular rate 143 beats per minute Stimulation rate 136 per minute The first five stimuli fall during the auricular refractory period and do not elicit premature beats The sixth stimulus, marked X, falls during a non-refractory period and an auricular premature systole results Arrows denote electrical stimuli

C Auricular paroxysmal tachycardia. The stimulation rate is 150 per minute and exceeds the rate of the basic sinus rhythm Each electrical stimulus is followed by an auricular and ventricular response The tracing is characteristic of auricular paroxysmal tachycardia

An injury current has been deliberately induced to make possible easy differentiation of the P and P′ waves from the electrical stimulation waves

ber of effective electrical stimuli relatively fewer; the basic cardiac rhythm remained under the influence of the sino-auricular node. At more rapid shock rates (usually above 150 stimuli per minute) the artificially stimulated ectopic focus generally became the pacemaker and auricular paroxysmal tachycardia supervened. The critical level at which the ectopic



A Auricular diastole.

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A shows auricle in complete diastole B shows contraction wave partially progressed *across* auricle with blood pooled *in the* distended appendix C [illegible] pleted. append[illegible]

focus usurped the pacemaking function depended upon the prevailing sinus rate; when the rate of stimulation exceeded that from the sinus node, the ectopic focus determined the basic cardiac rhythm (Figure 20).

Some workers have maintained that premature systoles can arise only *in the specialized* conduction system of the heart.[830] In this investigation no portion of the auricle subjected to an adequate stimulus failed to respond; premature systoles were produced at any site to *which a stimulus* was applied.

Three main sites of the right auricle were chosen for study: (1) the caudal border of the body near the inferior vena cava; (2) the tip of the appendix; and (3) the head of the sinoauricular node. On the left auricle two sites were chosen: (1) the appendix; and (2) the body of the auricle.

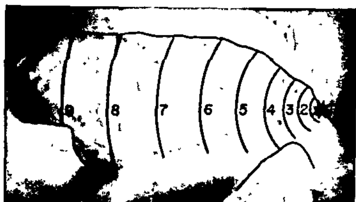
CINEMATOGRAPHIC APPEARANCE OF AURICULAR PREMATURE SYSTOLE IN THE EXPERIMENTAL ANIMAL

The premature beat begins as a wrinkling in the auricular wall in and around the ectopic focus. From *this focus* the wave of contraction spreads out *in all directions simultaneously*, and envelops the entire auricle which, with a puckering movement, is gathered in toward the focus in completion of systole. Diastole, which follows immediately, likewise begins at the ectopic focus and pursues a course identical *with that of systole* (Figure 21).

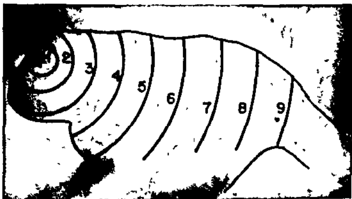
The sequence of events is the same regardless of the location of the ectopic focus. Figure 22 illustrates diagrammatically the course of the contraction wave of a auricular premature systole when the ectopic focus is (A) at the caudal end of the right auricle, (B) at the tip of the right auricular appendix; and (C) in the center of the body of the right auricle.

When the ectopic focus is located at the caudal end of the right auricle, *the* wave *is* seen to travel in a broad band across the length of the chamber, embracing its entire width from the auriculo-ventricular groove to the intercaval region (Figure 22A). When the ectopic focus is at the cephalic end of the auricle (tip of the appendix), the course of the wave is toward the caudal end (Figure 22B). When the ectopic focus is in the center of the auricle, the wave spreads outward from the focus in the circular fashion of ripples on a pond after a stone has been *dropped into it* (*Figure* 22C).

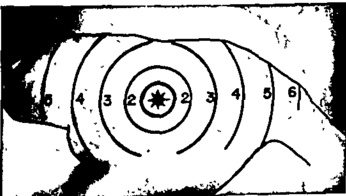
Premature contraction waves vary in



A. Focus at the caudal end of the auricle



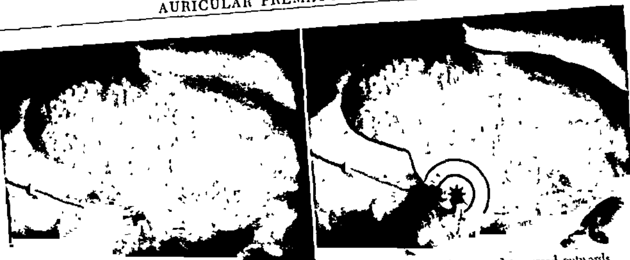
B Focus at the right appendix.



C Focus at the center of auricle

Figure 22. Stills with diagrammatic illustrations showing *the course of the contraction* waves of auricular premature systoles arising from differently placed ectopic foci (aconitine) on the right auricle

(A) Ectopic focus at the caudal end, (B) focus at the cephalic end of the right auricle, and *(C) focus in the center* Note that the contraction wave travels in all available directions



A. The head of the sinus node is electrically stimulated. The contraction wave has started to spread outwards



B The contraction wave has spread only a short distance from its origin at the head of the sinus node At this point, the contraction ended.



C Auricle now in complete diastole, following abortive contraction (A and B)

Figure 23 Example of incomplete contraction occasionally seen in cinematographs of auricular premature systoles Electrical impulse is introduced through a fine copper wire sutured to auricle at head of sinus node In A start of contraction wave is shown In B wave is seen to terminate after spreading over only a small part of the auricle and the contraction is at its maximum In C auricle has returned to complete diastole (compare with complete auricular premature beat in Figures 21 and 35)

ship to the cardiac cycle, and in degree of completeness.

ELECTROCARDIOGRAPHIC AND OSCILLOGRAPHIC APPEARANCE OF AURICULAR PREMATURE SYSTOLE

Observation 1: Shape of the P' Wave of Auricular Premature Systoles

With the discovery and widespread use of the electrocardiograph, certain characteristics of P' waves of auricular premature systoles have become well known. In experimentally produced auricular premature systoles in animals, in conventional limb and precordial leads the auricular deflections (P' waves) usually are found to differ in configuration from those of normal sinus beats (P waves);[178, 356] the P' waves are known to vary in shape with the location of the ectopic focus from which they originate. The P' waves inscribed by auricular premature systoles starting near the sinus node are similar to or identical with normal P waves and are recognizable only by their prematurity; such P' waves are generally upright in leads 1 and 2 (Figure 25A). P' waves of premature systoles starting at the caudal end of the auricle are deeply inverted, especially in leads 2 and 3 (Figure 25B).

Figure 26 shows auricular premature systoles recorded by the cathode-ray oscillograph from direct auricular leads in the dog. Minute details of the deflections, invisible in mechanically recorded tracings, are distinguishable. The waves of the premature beats are more complex and often wider than the normal. Frequently the P'-R interval is prolonged. The small, rapid components of the P' waves which appear in each complex are not artefacts; their exact signifi-

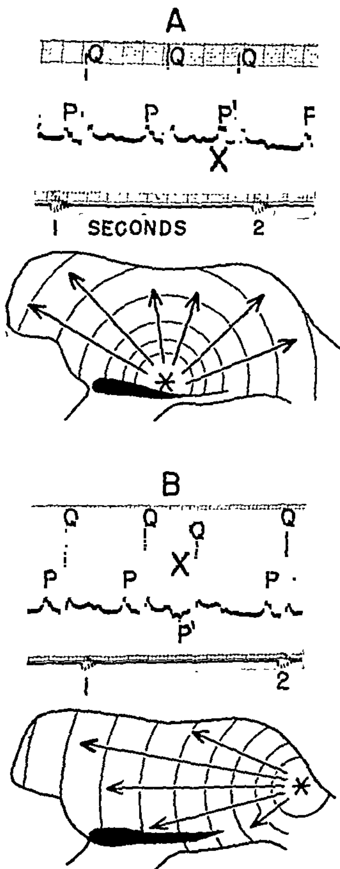


Figure 25 Premature auricular systole produced at various sites in right auricle of a dog Lead 2

(A) Auricular premature systole produced by mechanical stroking of right auricle near sino-auricular node. The 3rd beat is a premature auricular systole (X) starting near tail of the node Note that the shape of the P' wave is closely similar to that of the normal P wave

(B) Premature auricular systole produced by stroking at caudal end of auricle Note that the resulting P' wave is sharply inverted.

The direction of the P' wave in lead 3 is generally the same as that in lead 2. It is sharply inverted when the ectopic focus is produced at the caudal end of the auricle, it is upright or isoelectric when the impulse starts at or near the sinus node.

cance is unknown. Use of the oscillograph permits much greater accuracy in measurement of the P'-R interval than does the ordinary electrocardiograph.

Observation 2: Ta Wave Following Auricular Premature Systoles

Normally the P wave is not followed by a visible Ta wave (wave of repolarization); the latter is usually obscured by the succeeding ventricular complex.[1, 31, 159, 253, 337, 656] In auricular premature systoles, because of a delayed or blocked ventricular complex, a Ta wave may appear (Figure 27). The Ta wave assumes considerable importance in the high-frequency arrhythmias (Chapter VII).

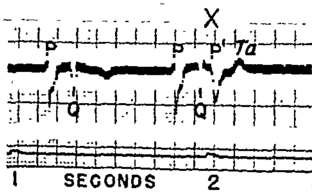


Figure 27. Normally the Ta wave which follows the P wave is invisible or masked by the ventricular complex. In this illustration (direct auricular lead) the third auricular wave (P') is an auricular premature systole produced by stroking the auricle. Because of the refractory state of the ventricle no ventricular complex follows; the Ta wave is easily seen. Because of the abnormal course of repolarization of the auricle during a premature systole, the Ta wave is abnormally large and prominent.

Observation 3: Ventricular Aberration in Auricular Premature Systole

Aberration of the ventricular complex may occur in all auricular arrhythmias. We have observed ventricular aberration after both experimentally produced and clinical auricular premature systoles (Figure 28). The distortion due to aberration affects the entire ventricular complex (QRS and T). Other workers believe that ventricular aberration in auricular premature systole is due to a disturbance of conduction tissue. Apparently the degree of aberration varies directly with the degree of prematurity;[6, 38, 521] however, other factors may also be related to the phenomenon. This subject is discussed in greater detail in Chapter XV.

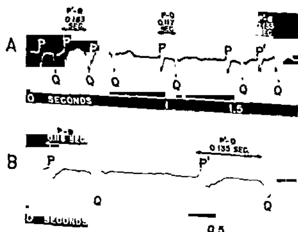


Figure 28. Direct auricular leads recorded with the oscilloscope.

(A) Three auricular premature beats (marked P') recorded at a relatively slow camera speed of approximately 6.5 cm per second. The P'-Q intervals are variable in duration and are longer than the normal.

(B) Two auricular beats taken at a camera speed of 19 cm. per second. There is one normal beat followed by one premature systole. The P' wave in this tracing is almost twice as wide as the normal P wave. The P'-Q interval can be studied in the oscillograph with a much greater degree of accuracy than hitherto possible. The duration of the P'-Q interval in seconds is marked on the illustration.

Observation 4: Course of the Excitation Wave of Auricular Premature Systole

In the following experiments, auricular premature systoles were produced at various sites on the right auricle; the course of the resulting excitation wave was studied by means of direct auricular leads recorded on a multiple-channel electrocardiograph and a dual-beam cathode-ray oscillograph. As noted in Chapter I, the intrinsic deflection is defined as the onset of the sharp negative wave inscribed as the cardiac impulse passes beneath the electrode. By recording two or three direct leads simultaneously, the times of arrival of a given impulse at various sites on the auricle could be compared to within approximately 0.01 second. Each experiment was repeated in at least four dogs and the accuracy of this type of study was

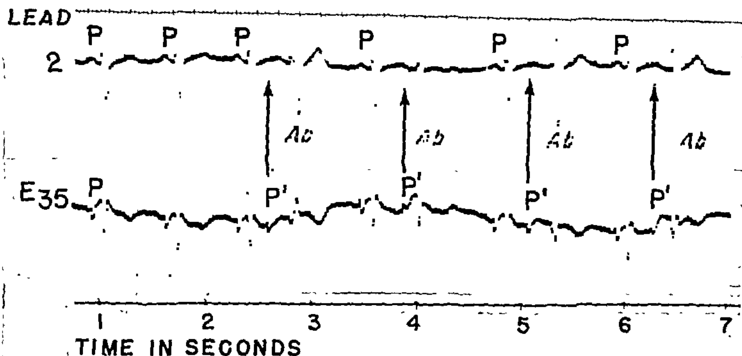


Figure 28. Simultaneously recorded lead 2 and esophageal lead from patient with auricular premature systole *In lead 2* no evidence is seen of auricular activity before the abnormal complex and it would probably be diagnosed as nodal or ventricular premature systoles In the simultaneously recorded esophageal lead (E 35) a P′ wave can be seen before each aberrant QRS complex Therefore, the diagnosis is auricular premature systoles with ventricular aberration.

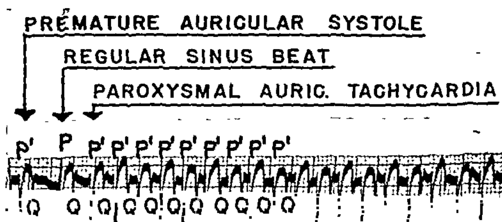


Figure 29. Premature auricular systole and paroxysmal auricular tachycardia produced by *mechanical stroking of right auricle in dog* Direct auricular lead. First complex is a premature auricular systole, second complex is a normal sinus beat After 3rd beat there is a paroxysm of auricular tachycardia Note that the P′ wave in the auricular premature beat and the P′ waves in the paroxysm of auricular tachycardia are identical in shape The P wave of the normal sinus beat is of different shape

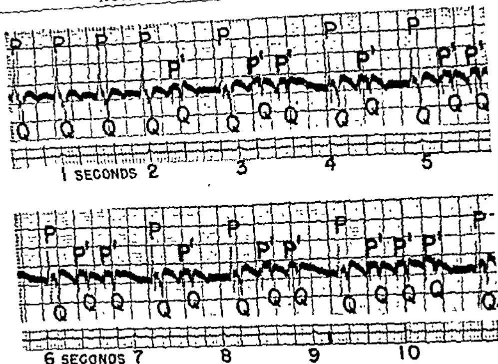


Figure 30. Isolated auricular premature systoles and short paroxysms of auricular tachycardia from an ectopic focus produced by aconitine at the caudal end of the right auricle in a dog Upper and lower strips represent one continuous tracing from a direct auricular lead. All complexes marked P are of regular sinus origin The auricular complexes markd P' are premature auricular beats Of these the 5th and 10th beats on the
on the lower
in "runs" of
Note that the
beats are

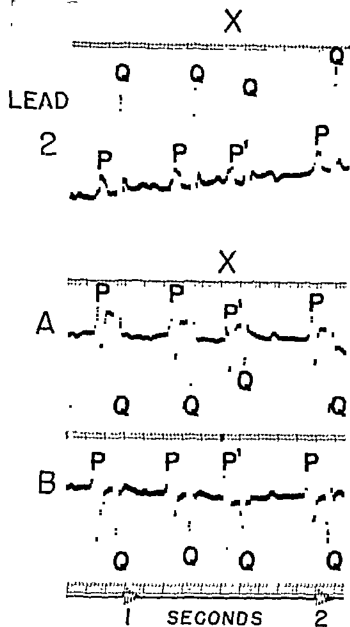
greatly increased by use of the cathode-ray oscillograph

Occasionally, when an auricular premature systole was produced at a certain site, "runs" of such premature beats (auricular tachycardia) resulted; more rarely, auricular flutter and fibrillation were obtained (Figures 29 and 30).

Focus at Tip of Right Appendix: Two nonpolarizable electrodes [365] (described in the Appendix) were placed on the right auricular wall on a line perpendicular to the sulcus terminalis (Figure 31) Electrode A was adjacent to the auriculo-ventricular groove and electrode B was just above the sulcus terminalis. Premature systoles were produced at the tip of the right auricular appendix at a point approximately equidistant from the two electrodes by mechanical stroking of the area with a wooden applicator stick.

Figure 31 shows that the intrinsic deflections from both electrodes were inscribed simultaneously during the premature systoles. Hence, the electrical impulse from the ectopic focus at the tip of the right auricular appendix must have arrived at both electrodes simultaneously. When the experiment was repeated with the ectopic focus located at the caudal end of the right auricle, equidistant from the two electrodes, the impulse again arrived simultaneously at both electrodes (Figure 32). Since the electrodes were placed to include in their span the full width of the body of the auricle, it may be concluded that the excitation wave of auricular premature systole travels from the ectopic focus across the auricle in a broad band and involves the full width of that chamber.

Focus at Caudal End of Right Auricle: A second experiment was performed with two electrodes placed on the auricle in a line *parallel* to the sulcus terminalis, between it and the



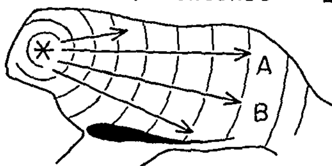
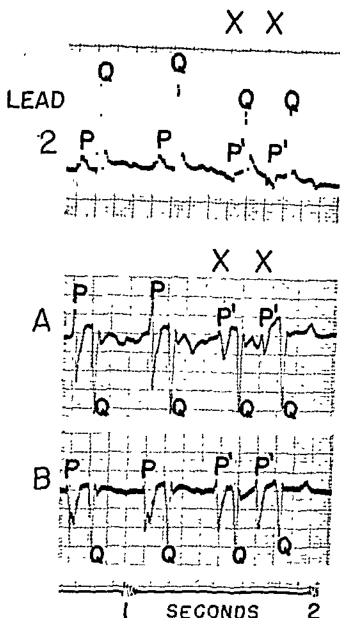


Figure 31 Electrocardiograms of an auricular premature systole produced at the tip of the right appendix in a dog by mechanical stroking. Lead 2 and direct auricular leads A and B were recorded simultaneously. Electrode A is near the auriculo-ventricular groove, B near the sulcus terminalis (each electrode equidistant from the ectopic focus) During normal sinus rhythm the intrinsic deflection of the P wave in B (which is closer to the sino-auricular node than A) is ahead of that of A by about 0 02 second Note also that B has a smaller upright auricular deflection than A, thus indicating that the normal impulse reaches B before A The 3rd beat is the auricular premature beat In this instance, the intrinsic deflections occur simultaneously, and confirms the fact that the ectopic impulse reached A and B at the same time. Observe that the P′ wave in lead 2 is similar to the normal P wave Note also that the intrinsic deflecions of the normal and premature beat occur during the P and P′ waves in lead 2



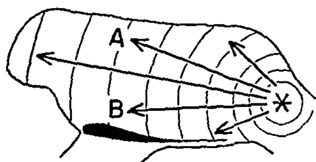


Figure 32 Electrocardiograms of auricular premature systoles produced at the caudal end of the right auricle in a dog by mechanical stroking The 3rd and 4th beats (X) are auricular premature systoles Lead 2 and direct auricular leads were recorded simultaneously Electrode A is near the auriculo-ventricular groove, B near the sino-auricular node (each electrode equidistant from the ectopic focus). The intrinsic deflection of the P wave of B in the normal beats is about 0 02 second ahead of A The intrinsic deflection in B is almost entirely negative, whereas in A, it is large and positive Thus it is indicated that in normal sinus rhythm the impulse travels from B to A The intrinsic deflections of the two premature beats occur at exactly the same instant in A and B, respectively The P′ wave in lead 2 is deeply inverted in contradistinction to the upright P′ wave of lead 2 in Figure 31 This is due to the difference in the location of the ectopic focus in the two experiments

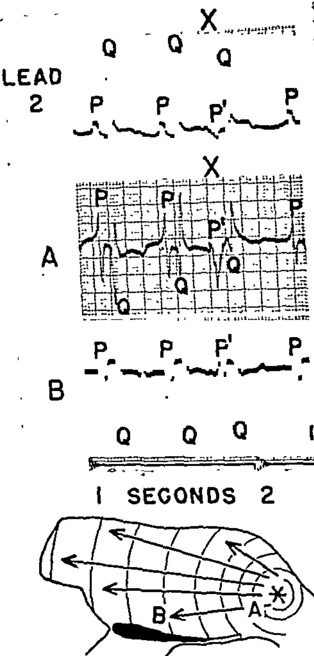


Figure 33 Demonstration that the impulse originating at the caudal end of the right auricle travels in a direction different from that of the normal sinus beat Lead 2 and direct auricular leads were recorded simultaneously Electrode A is near the site of ectopic focus (caudal end of auricle), B is near the sino-auricular node (site of origin of normal sinus beat) The 3rd beat is the premature systole produced by mechanical stroking

In the first two (normal) beats there is a pure negative deflection in the P wave in B and a positive deflection in A The intrinsic deflection of the P wave in B occurs approximately 0 04 second ahead of A This indicates that in regular sinus rhythm the impulse travels from B towards A In the premature systole (3rd beat) the more positive deflection occurs in B, the intrinsic deflection in A is now ahead of B. The P' wave in lead 2 is inverted. Thus the impulse of a premature auricular systole starting at the caudal end of the auricle travels in a caudocephalic direction, a course different from that of the normal sinus beat

auriculo-ventricular groove. An ectopic focus was established by mechanical stimulation at the caudal end of the right auricle (Figure 33).

The intrinsic deflections reveal that the impulse reached electrode A before electrode B during the premature systole. Thus, it was demonstrated that the excitation wave traveled from the site of origin at the caudal end of the right auricle across the chamber in a caudo-cephalic direction.

Focus at Center of Right Auricular Body: Two electrodes, one dorsal (B) and one ventral (A), were placed on the body of the right auricle equidistant (approximately ½ centimeter) from the site to be stimulated (Figure 34). Premature systoles were produced by mechanical stroking at the center of the body of the auricle.

During normal sinus beats, the intrinsic deflections from electrode B were inscribed approximately 0.03 second before those from electrode A (Figure 34). Thus, the excitation wave of the normal sinus beat traveled in a ventral direction from the sinus node toward the periphery. When the premature beat was produced at the ectopic focus in the center of the auricle, the intrinsic deflections from electrodes A and B were inscribed simultaneously, indicating that the excitation wave spread from the focus of origin in dorsal and ventral directions simultaneously and at the same rate of propagation.

Summary: The tracings of auricular premature systoles obtained in the above experiments show that the impulse leaves the ectopic site of origin and spreads outward over the auricle in all available directions simultaneously. It is thus demonstrated that *in premature auricular systole the course of the excitation wave as determined by direct lead electrocardiography corresponds to the course of the contraction wave from the same site as visualized by high-speed cinematography* (Figures 21, 22 and 35).

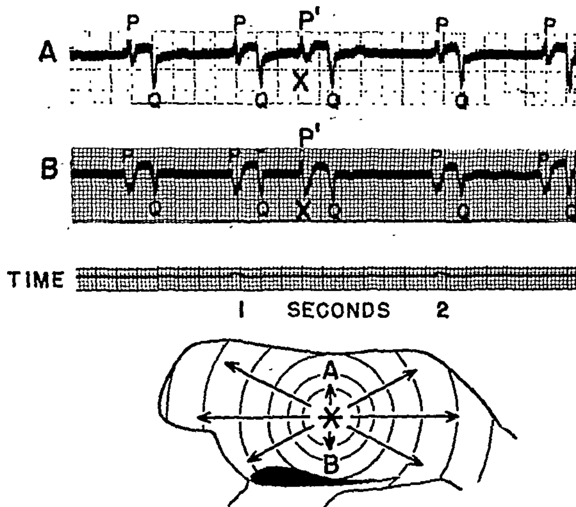


Figure 34 Course of the impulse from an ectopic beat arising in the center of the auricle, relation to course of normal sinus impulse Direct auricular leads were recorded simultanously Electrode A is near the auriculo-ventricular groove, electrode B near sino-auricular node (each equidistant from ectopic focus). During normal sinus beats, the intrinsic deflections of the P waves of electrode B were inscribed about 0.03 second before A. This demonstrates that the normal sinus excitation wave traveled in a ventral direction from the sinus node toward the periphery When the premature beat was produced the intrinsic deflections from electrodes A and B were inscribed simultaneously, indicating that the excitation wave spread from the focus of origin in dorsal and ventral directions at the same time and at the same rate of propagation

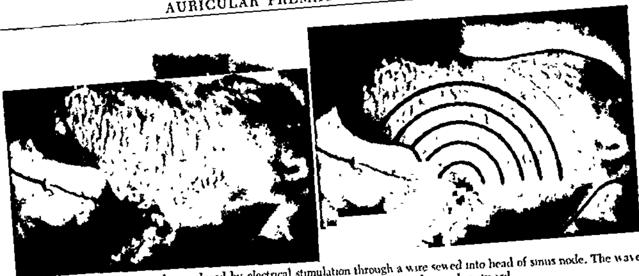
EFFICIENCY OF AURICULAR PREMATURE SYSTOLES

The successive occurrence of auricular premature systoles and normal sinus beats as visualized on cinematographs makes possible an accurate comparison of the relative efficiency of these two cardiac rhythms. It has been observed consistently that premature contractions are less efficient than normal beats.

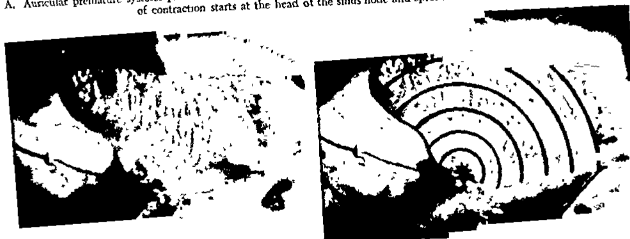
The efficiency of a given premature contraction depends upon three factors: (1) the time relationship of the beat to the cardiac cycle (degree of prematurity); (2) the distance of the ectopic focus from the normal sinus pacemaker; and (3) the degree of completeness of the beat.

(1) In general, the earlier in diastole a premature contraction arises, the less efficient it will be. In the initial phases of diastole the auricle is only partially filled and but a small amount of blood is available for transfer to the ventricle, later in diastole auricular filling is more complete and the supply of blood for systole more adequate.

(2) The greater the distance of the ectopic focus from the normal sinus pacemaker, the less efficient is the premature beat. The cinematographs showed that beats arising from foci on



A. Auricular premature systoles produced by electrical stimulation through a wire sewed into head of sinus node. The wave of contraction starts at the head of the sinus node and spreads outward



B The cont



C The appendix and body of the auricle are now in diastole, but the caudal portion of the auricle is now contracted This forces blood back into the appendix and body of the auricle Note distention of appendix.

Figure 35 Auricular premature systole (produced by electrical stimulation) starting at head of sino-auricular node As made clear by diagrammatic illustrations the premature contraction wave reaches the cephalic before the caudal end of the auricle. This results in asymmetrical auricular contractions, such contractions are obviously less complete, hence less efficient.

the right auricle furtherest from the tail of the sinus node, such as the extreme caudal end of the right auricle (Figure 21), were most inefficient; the contraction wave had to travel approximately twice as far as the normal wave to reach remote parts of the chamber. In its course, the wave propelled the auricular contents toward the right auricular appendix where blood was pooled as in a blind pouch. When on completion of systole the contraction wave enveloped the auricular appendix, the pooled blood was displaced backward to the caudal end of the auricle in which region diastole had already begun. Thus blood was shuttled from one end of the auricle to another with only small volumes being funneled through the tricuspid orifice into the right ventricle. These events are demonstrated in Figure 21. In Figure 21B the auricular premature systole has covered part of the auricle and the appendix is distended with pooled blood. In Figure 21C the premature systole is completed, the appendix is fully contracted, and the pooled blood is displaced backward into the relaxed caudal end of the auricle.

(3) The observation has been made repeatedly that the contraction wave of auricular premature systole frequently spends itself before reaching the periphery of the auricle. Under such circumstances, auricular emptying is only partially effected and to that extent such systole is obviously inefficient.

Premature beats arising near the tail of the sinus node (close to the normal pacemaker) are more efficient than those from the head of the node. In the former, the effectiveness of the contractions is impaired only by prematurity and incompleteness of systole; the adverse effect of distance from the normal pacemaker is eliminated. In the latter instances, the head of the node is sufficiently far removed from the normal pacemaker (Chapter I) to make the factor of distance operative (Figure 35)

Clinical Correlation: The time relationship of the premature beat to the cardiac cycle may serve to explain two clinical phenomena: (1) During some auricular premature systoles only the first heart sound is heard. When a premature beat occurs early in diastole the amount of blood transferred to the ventricles may be too small to open the semi-lunar valves so that the second heart sound is not audible. (2) Occasionally an auricular premature systole is associated with subjective sensations in the jugular areas. When an auricular premature beat occurs simultaneously with ventricular systole, the tricuspid valve is closed and serves as a dam behind which a large amount of blood collects in the jugular veins. This trapped blood may distend the veins and cause subjective symptoms.

Visual Confirmation of Starling's Law: Since the premature beat is less efficient than the normal beat, it usually permits a large volume of residual blood to remain in the auricle. This results in greater diastolic filling immediately preceding the next regular sinus beat. The cinematographs reveal that the succeeding contraction — the first sinus beat after the premature beat — is more virogous and complete than the normal systole. The greater intensity of this contraction is undoubtedly a direct result of the greater volume of blood in the preceding diastole and is thus a visual confirmation of Starling's Law, namely, that the force of the systolic contraction is proportional to the degree of diastolic filling (see Chapter I). This increased load after the pause may account for the transient pulsus alternans frequently noted clinically immediately following premature systoles.

DISCUSSION

The fact that the experimental production of auricular premature systoles depends upon a specific relationship between the rate of stimulation at the ectopic focus and the prevailing sinus rate suggests an explanation for certain well recognized clinical aspects of this arrhythmia. Clinically, premature systoles are apt to occur when the basic sinus rhythm is slow, and premature beats may be eliminated when the basic sinus rate is accelerated. A common bedside method of differentiating multiple prema-

ture beats from auricular fibrillation is the employment of measures which either slow or accelerate the cardiac rate. When the rate is slowed, the rhythm of auricular fibrillation tends to become less irregular, whereas that of multiple premature systoles becomes more irregular due to an increase in the number of premature beats. When the rate is accelerated, the results are usually reversed The influence of the basic auricular rate on the occurrence of premature beats is due to variations in length of refractory periods. When the rate is slow, the nonrefractory phase between beats is relatively long and stimuli at an ectopic focus can readily contact excitable muscle to cause premature contractions When the basic rate is rapid, the nonrefractory period is short and there is little opportunity for stimuli at the ectopic site to take effect. For the same reason, because of the extremely rapid basic rates in auricular paroxysmal tachycardia, auricular flutter and auricular fibrillation, stimuli from other ectopic foci which might cause premature systoles during normal sinus rhythm cannot become effective. Hence, no premature auricular contractions occur during these rapid rate arrhythmias.

SUMMARY AND CONCLUSION

The contraction wave of experimentally produced auricular premature systole in the dog has been observed by means of high-speed cinematography; the associated electrical excitation wave has been studied by direct and indirect lead electrocardiography and cathode-ray oscillography. The course of the contraction wave as observed in the motion pictures is identical with the course of the excitation wave as seen in the electrocardiograms and oscillograms.

Auricular premature systoles were experimentally produced in two human subjects by mechanical stimulation of the auricle during surgical procedures in the chest. The contraction and relaxation waves of the arrhythmia in man are identical with those in the dog.

Both photographically and electrically, the auricular premature systole and the normal sinus beat are essentially analogous. In each rhythm the contraction and excitation waves arise from a single specific site, the normal beat from the sino-auricular node, the premature beat from an ectopic focus. In both rhythms each contraction and relaxation wave spreads out from the site of origin in an identical manner, travelling in all available directions simultaneously. The auricular premature systole differs from the normal beat in location of the site of origin, in time relationship to the cardiac cycle (factor of prematurity), and in completeness of contraction. In direct relation to the extent to which a premature systole differs from normal with respect to these factors, its efficiency is adversely affected.

CHAPTER III

Auricular Paroxysmal Tachycardia

CLINICAL CONSIDERATIONS

Auricular paroxysmal tachycardia is a condition characterized by repeated bouts of regular heart action at rates faster than normal sinus rhythm. The tachycardia rate range is usually between 140 and 250 beats per minute, most often between 160 and 200 beats per minute. In most instances duration of an attack is from a few minutes to several hours, although the less common paroxysm persists for days, weeks or even months. The onset of the arrhythmia with all its attendant clinical phenomena is sudden and startling; the transition back to normal sinus rhythm with complete relief of symptoms is equally abrupt. The onset and termination of the attacks each occurs within a single cardiac cycle. Not infrequently isolated premature auricular systoles precede and follow paroxysms of tachycardia.

The nature and mechanism of auricular paroxysmal tachycardia are discussed in detail later in this Chapter.

Etiologic and Pathologic Factors: No specific etiologic factors or pathologic lesions have been identified in this disturbance. Like auricular premature systole, auricular paroxysmal tachycardia occurs often in otherwise normal hearts and must therefore be regarded as essentially functional in origin. When it occurs in diseased hearts there is no constant association with any specific lesion. Although the arrhythmia apparently is somewhat more frequent in mitral stenosis, it may be seen in all other types of valvular lesions, in myocardial damage without valve lesions, and in certain types of congenital heart disease including auricular septal defects, Eisenmenger complex, downward displacement of the tricuspid valve, drainage of all pulmonary veins into the right heart, and isolated dextrocardia. It may appear as the first sign of coronary artery disease and on occasion it follows myocardial infarction.

Age and Sex Incidence: Males and females are affected with equal frequency. The arrhythmia is rare in infancy and early childhood and becomes increasingly common after adolescence; its incidence is greatest in the third and fourth decades. In infants, the arrhythmia is often serious and may prove fatal; not infrequently it occurs in association with the Wolff-Parkinson-White syndrome. In the younger age group (first four decades), paroxysms occur most frequently among normal persons or patients with rheumatic heart disease or hyperthyroidism. In age groups beyond the fourth decade the incidence of the arrhythmia is greatest among subjects with myocardial disease and hypertension; it is less prevalent in older persons with rheumatic heart disease or hyperthyroidism

Precipitating Factors: In persons subject to this arrhythmia attacks often occur under the following circumstances. (1) emotional disturbances, (2) unusual effort or exertion, (3) acute digestive disorders (flatulence, etc.), and (4) sudden changes in posture

The arrhythmia may be initiated during or immediately after surgical operations. Paroxysms are not uncommon during deep anesthesia or during the first 24 hours postoperatively, these attacks may be related to hypoxia. Bouts of tachycardia have been reported to result from endotracheal intubation during anesthesia;

these instances were attributed to a reflex mechanism. A similar mechanism was suggested for auricular tachycardia accompanying manipulations of various structures in the thorax (pericardium, pleura, etc.). Cardiac catheterization may induce auricular paroxysmal tachycardia, although auricular premature systoles are a more common complication of that procedure; the resulting paroxysms usually are of brief duration and no serious effects have been noted. A number of instances of auricular paroxysmal tachycardia have been observed following digitalis medication, either as a result of toxic overdose or as a true idiosyncrasy.[278] The arrhythmia is an infrequent complication of postural hypotension, in such instances the disturbance appears when the patient stands and may result in severe disability. One patient has been seen in whom either premature systoles, fibrillation or tachycardia developed whenever an upright position was assumed. Paroxysms may accompany gastro-intestinal disorders or gallbladder disease. The arrhythmia may occur during pregnancy. Many patients are awakened from sleep by tachycardia, presumably the disturbance is induced by depression of the sino-auricular node or by the excitement of disturbing dreams. Exercise and excitement appear to be among the most important precipitating factors. Within a period of three months we have seen four patients in whom paroxysms could be induced by exercise.

A number of interesting and unusual cases have been recorded in the literature. An instance of nodal tachycardia occurring postpartum is reported;[434] the arrhythmia was accompanied by severe shock, but the patient responded well to therapy. Auricular paroxysmal tachycardia was noted in two patients with cervical ribs,[533] apparently a direct causal relationship existed, since the arrhythmia disappeared following resection of the ribs. One case of auricular paroxysmal tachycardia was found associated with hypothyroidism.[532] Two instances are described in which paroxysms of supraventricular tachycardia almost invariably occurred immediately preceding each menstrual cycle;[118] in one instance the attacks began at puberty, in the other later in life. A case is reported in which the patient was able to induce a paroxysm by holding his breath and tightening his abdominal muscles (Valsalva maneuver).[201]

Symptomatology: The symptoms of auricular paroxysmal tachycardia vary with the cardiac rate, the duration of the paroxysm and the underlying state of the heart. When the rate is moderate and the paroxysm of short duration few or no subjective symptoms may prevail; the patient is aware only of a transient "fluttering" sensation in the chest which upon reassurance he learns to ignore. When the paroxysm lasts an hour or longer it may cause dyspnea, anxiety, and sometimes angina-like pain. With the progression of the attack these symptoms usually become aggravated and more severe signs of congestive failure occasionally develop. Syncope is not an uncommon complication either at the onset or at any time during the course of the attack. In the presence of coronary artery disease, severe and sometimes prolonged anginal pain (status anginosus) may occur. Although all these complications are more apt to develop in the presence of significant myocardial disease, prolonged paroxysms of tachycardia at rapid rates may precipitate serious congestive failure even in otherwise normal hearts This is especially true in infants in whom the arrhythmia not infrequently terminates fatally.

In the great majority of instances the paroxysms end abruptly with rapid disappearance of all symptoms. Occasionally the termination is marked by a sudden thumping of the heart associated with a stabbing pain in the chest; these events are fleeting in duration and are followed rapidly by a sensation of complete relief. With the termination of the paroxysm all physical signs rapidly recede. After a prolonged attack, however, residuals may persist for several hours.

Diagnosis: The diagnosis in most instances presents little difficulty and can usually be established at the bedside. The distinguishing features are the sudden onset and a rate

CHAPTER III

Auricular Paroxysmal Tachycardia

CLINICAL CONSIDERATIONS

AURICULAR paroxysmal tachycardia is a condition characterized by repeated bouts of regular heart action at rates faster than normal sinus rhythm. The tachycardia rate range is usually between 140 and 250 beats per minute, most often between 160 and 200 beats per minute. In most instances duration of an attack is from a few minutes to several hours, although the less common paroxysm persists for days, weeks or even months. The onset of the arrhythmia with all its attendant clinical phenomena is sudden and startling, the transition back to normal sinus rhythm with complete relief of symptoms is equally abrupt. The onset and termination of the attacks each occurs within a single cardiac cycle. Not infrequently isolated premature auricular systoles precede and follow paroxysms of tachycardia.

The nature and mechanism of auricular paroxysmal tachycardia are discussed in detail later in this Chapter

Etiologic and Pathologic Factors: No specific etiologic factors or pathologic lesions have been identified in this disturbance. Like auricular premature systole, auricular paroxysmal tachycardia occurs often in otherwise normal hearts and must therefore be regarded as essentially functional in origin. When it occurs in diseased hearts there is no constant association with any specific lesion. Although the arrhythmia apparently is somewhat more frequent in mitral stenosis, it may be seen in all other types of valvular lesions, in myocardial damage without valve lesions, and in certain types of congenital heart disease including auricular septal defects, Eisenmenger complex, downward displacement of the tricuspid valve, drainage of all pulmonary veins into the right heart, and isolated dextrocardia. It may appear as the first sign of coronary artery disease and on occasion it follows myocardial infarction.

Age and Sex Incidence: Males and females are affected with equal frequency. The arrhythmia is rare in infancy and early childhood and becomes increasingly common after adolescence; its incidence is greatest in the third and fourth decades. In infants, the arrhythmia is often serious and may prove fatal; not infrequently it occurs in association with the Wolff-Parkinson-White syndrome. In the younger age group (first four decades), paroxysms occur most frequently among normal persons or patients with rheumatic heart disease or hyperthyroidism. In age groups beyond the fourth decade the incidence of the arrhythmia is greatest among subjects with myocardial disease and hypertension; it is less prevalent in older persons with rheumatic heart disease or hyperthyroidism.

Precipitating Factors: In persons subject to this arrhythmia attacks often occur under the following circumstances: (1) emotional disturbances; (2) unusual effort or exertion; (3) acute digestive disorders (flatulence, etc.); and (4) sudden changes in posture

The arrhythmia may be initiated during or immediately after surgical operations Paroxysms are not uncommon during deep anesthesia or during the first 24 hours postoperatively; these attacks may be related to hypoxia. Bouts of tachycardia have been reported to result from endotracheal intubation during anesthesia;

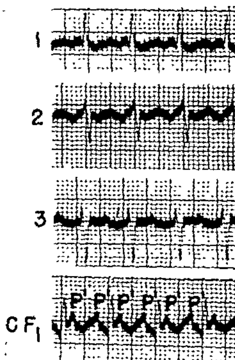


Figure 39 Auricular flutter with 2 1 auriculo-ventricular block. Standard limb leads 1, 2 and 3 simulate auricular paroxysmal tachycardia, but in the chest lead the diagnosis of flutter is apparent Evans[44] recently emphasized this phenomenon

(usually between 160 and 200 beats per minute) which remains absolutely constant and perfectly regular throughout the paroxysm. Upon auscultation the sounds are all alike in quality and intensity. Maneuvers which tend to increase vagal tone (carotid sinus pressure, holding of breath, etc) either leave the rate and rhythm totally unaltered or result in a sudden and complete change to normal slow sinus rhythm. These features are possessed by no other arrhythmia and are therefore diagnostic A further detailed differential diagnosis of the regular tachycardias is presented in the chapter on auricular flutter (Table I, Chapter V).

The electrocardiographic diagnosis is usually definitive The P′ wave of tachycardia may be deeply inverted, diphasic or notched, frequently it is similar to the P wave of normal sinus rhythm which is upright (Figures 36, 37 and 38).

Several sources of error in electrocardiographic diagnosis are worthy of note:

(1) In standard limb leads, flutter may simulate tachycardia when alternate flutter waves are buried in the ventricular complexes (Figure 39). This problem is discussed in a recent paper by Evans.[176]

(2) When the P′ wave of auricular tachycardia is buried in the ventricular complex the condition may be diagnosed as nodal tachycardia (Figure 40) or, in the presence of bundle branch block, the differential diagnosis between auricular and ventricular tachycardia may become difficult or impossible (Figure 41).

(3) In the presence of partial or varying auriculo-ventricular block, auricular paroxysmal tachycardia may simulate auricular fibrillation in leads with P′ waves of low amplitude.

(4) Unusually rapid sinus tachycardia (rate over 150 beats per minute) may be confused

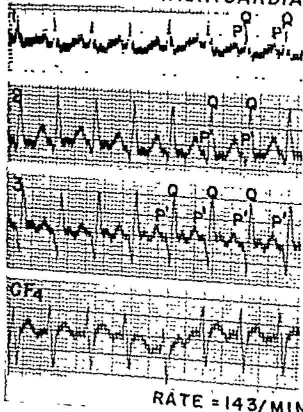


Figure 40. Electrocardiogram from a patient with auricular paroxysmal tachycardia simulating nodal tachycardia. The P′ waves are superimposed on the last portion of the T waves; as a result the T waves appear large and upright, and the Q-T intervals appear falsely prolonged.

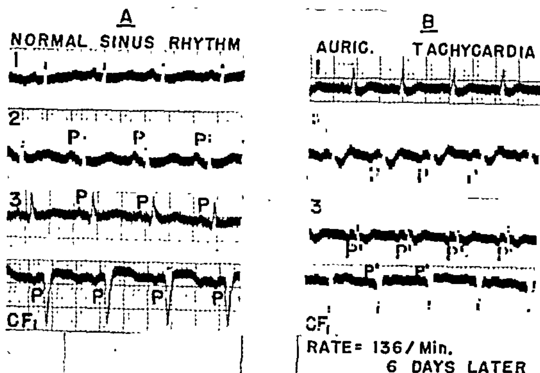


Figure 36 Electrocardiograms of a patient taken during (A) normal sinus rhythm and (B) during an attack of auricular paroxysmal tachycardia which occurred six days later
(A)
(B)

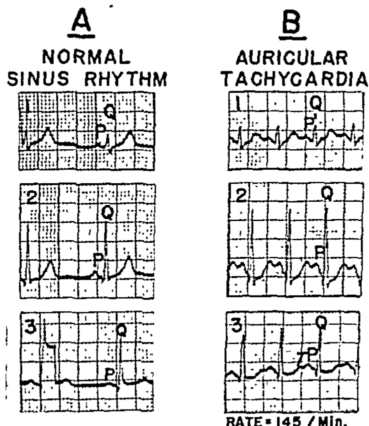


Figure 37 Electrocardiogram of a patient taken (A) before and (B) during a paroxysm of auricular tachycardia *The P and P' waves in leads 1, 2 and 3 are similar in both A and B*

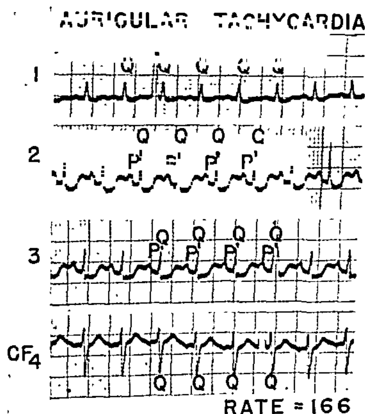


Figure 38 Electrocardiogram of patient with auricular paroxysmal tachycardia The P' waves are upright in leads 1, 2 and 3 *Auricular rate is 166 beats per minute*

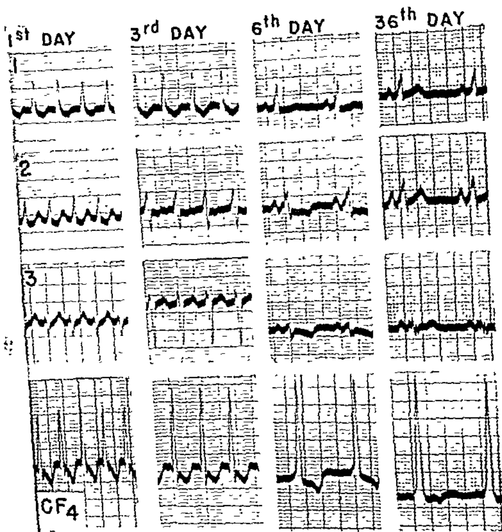


Figure 42 Auricular paroxysmal tachycardia producing electrocardiographic changes of myocardial fatigue (coronary insufficiency) in a 56 year old male with arteriosclerotic heart disease and the Wolff-Parkinson-White syndrome

1st day of auricular tachycardia S-T depression in leads 1, 2 and CF_4 and diphasic T in lead 2, and inverted T in CF_4.

3rd day of auricular tachycardia S-T depression in leads 1, 2 and CF
T waves in leads 1 more deeply inverted T

6th day Normal sinus rhythm resto
and has newly appeared in lead 3. The T
right in lead 1, but has become inverted i

36th day With the exception of the inverted T waves in CF_4, the signs of coronary insufficiency have almost completely disappeared

With the restoration of normal sinus rhythm (since 6th day) the characteristic pattern of the Wolff-Parkinson-White syndrome has returned

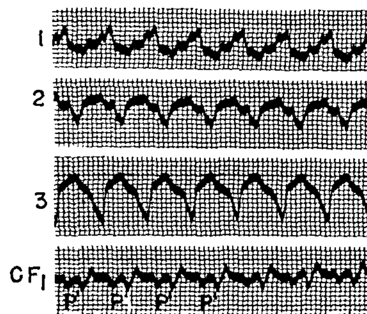


Figure 41. Auricular tachycardia with left bundle branch block simulating ventricular tachycardia. Leads 1, 2 and 3 exhibit no definite evidence of auricular activity and suggest a diagnosis of ventricular tachycardia. In the chest lead, however, inverted P' waves appear before each ventricular complex, definitely establishing the presence of auricular tachycardia.

with auricular paroxysmal tachycardia. Sedation and carotid sinus pressure are helpful in differential diagnosis. Sedation usually causes gradual slowing of the rate in sinus tachycardia but not in paroxysmal tachycardia. Carotid sinus pressure slows the rate of sinus tachycardia slightly while pressure is maintained; in auricular paroxysmal tachycardia this procedure either fails to affect the rate or suddenly terminates the arrhythmia.

These problems in interpretation sometimes can be eliminated through the use of esophageal leads and high precordial (V and CR leads). The increased amplitude of the P' waves recorded in such leads makes the auricular complexes more distinct (Figures 39 and 41).

Because of the marked difference in prognosis and treatment, the importance of distinguishing ventricular from auricular tachycardia is apparent. When the P' waves of the electrocardiogram and the "a" waves of the jugular pulse occur at a rate slower than the ventricular rate, the tachycardia is of ventricular origin. If ventricular tachycardia occurs without retrograde block, however, a differential diagnosis may become difficult. Other types of tachycardia are discussed later in this chapter.

Prognosis: In general, the prognosis for most patients with auricular paroxysmal tachycardia is favorable. The majority of attacks occur in persons with otherwise normal hearts; in at least half the cases the paroxysms terminate either spontaneously after a brief period or can be terminated readily by simple means such as carotid sinus or ocular pressure.

In patients with structural heart disease, especially coronary artery disease, attacks of rapid-rate tachycardia which are resistant to therapy may precipitate serious complications, namely, congestive failure, prolonged anginal pain, collapse and death. As a result of rapid heart action the effective coronary blood flow may be decreased while the work of the heart is greatly increased. Subendocardial and more diffuse myocardial necrosis has been observed under such circumstances. Appearance of electrocardiographic evidence of myocardial ischemia during or after paroxysms of tachycardia is common (Figure 42). Under such conditions auricular paroxysmal tachycardia assumes great prognostic importance. Sometimes even the most careful management fails to terminate the arrhythmia and the patient succumbs.

More rarely, in the absence of structural heart disease, rapid-rate tachycardias which persist for many hours, days or weeks may lead to similar serious complications. This is strikingly illustrated in tachycardia in infants in whom the rate is apt to be extremely rapid and the paroxysm prolonged and resistant to therapy. Even in adults without structural heart disease, serious complications occasionally occur. An interesting case of this type is reported by Levine.[345] "A man of 40 who was otherwise perfectly well had had three attacks of tachycardia during the previous few years. Each attack lasted uninterruptedly for five to 11 days. During the first one he developed a hemiplegia from which he gradually recovered in the course of a few months with a slight residual spasticity of one side of the body. During the

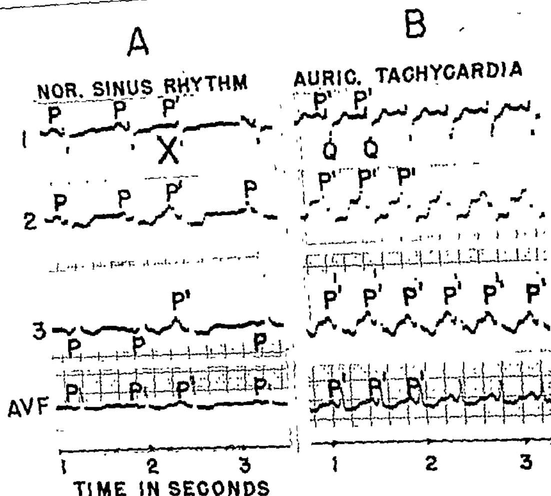


Figure 43 Electrocardiograms of patient having rheumatic heart disease associated with frequent episodes of auricular premature systoles and auricular paroxysmal tachycardia

(A) Sinus rhythm The 3rd beat is an auricular premature systole with large upright P' waves.

(B) A tracing taken a few seconds after A shows auricular paroxysmal tachycardia. Note that the P' waves of the tachycardia are of the same configuration as the P' wave of the auricular premature systole in A.

EXPERIMENTAL STUDIES

In spite of its importance, the mechanism of auricular paroxysmal tachycardia has received comparatively little attention from investigators. As a result, opinions concerning its nature are diverse and facts are few.

While the writings of ancient authors probably include accounts of this dramatic ailment, in modern times the earliest descriptions of auricular paroxysmal tachycardia are those of Cotton in 1867,[113, 114] Bristowe in 1887,[70] and Bouveret in 1889,[53] Bouveret named the condition "tachycardia paroxystique essentitielle." Herringham in 1897[234] credited Tuchzek, in 1878, with the suggestion that vagus paralysis was responsible for the arrhythmia, and advanced the rival theory of "irritation of the sympathetics."

On the basis of a study of circulating excitations in rings cut from turtle hearts, Mines[438] proposed that a circus movement might be causal in some instances of clinical paroxysmal tachycardia. Observations on the effects of quinidine led Illiescu and Sebastiani[293] to a similar conclusion. Ashman and Hull[6] in 1944 expressed the opinion that paroxysmal tachycardias were often, though not always, due to a circus movement into and out of the sino-

second attack he developed aphasia which gradually disappeared in four months. During the third attack, dry gangrene of the left arm developed requiring amputation at the shoulder. We saw this man in 1914 during the fourth attack and found the heart rate to be 250 a minute. After obtaining certain data on this patient the attack was immediately ended by ocular pressure. It was the first time his attacks had been controlled, for the others had stopped spontaneously after lasting many days. In fact this may have been the first instance in which ocular pressure was ever effective as a treatment for paroxysmal tachycardia." In explanation of the complications that occurred in this case Levine points out that "while the heart was beating 250 times a minute the pulse pressure was extremely low. During several attacks in which the patient was observed the systolic pressure was around 94 to 96 and the diastolic around 88 mm. Therefore, this patient had an effective pulse pressure of no more than 6 or 8 mm. One can readily see from this how thrombosis in peripheral vessels could easily develop. This must have occurred in the cerebral vessels during two of the attacks and in the vessels of the left arm at the time gangrene occurred. The process essentially consisted of stagnation of the blood. In such a case it is evident that proper therapy was imperative, for this patient is still alive and in fairly good health"

Treatment: The effectiveness of prophylaxis and treatment varies considerably. Prophylaxis consists of elimination of possible aggravating factors, correction of pathologic states which tend to precipitate the arrhythmia, and administration of indicated drugs

All aggravating factors such as excitement, fatigue, and excessive use of tobacco, alcohol and coffee should be eliminated. Adequate oxygen should be supplied during surgical procedures and the postoperative period of respiratory depression. Abnormalities of blood pressure or thyroid function should be controlled. Cervical ribs may be resected. Paroxysms of tachycardia associated with gastrointestinal dysfunction may be prevented by suitable diet and antispasmodics; removal of a diseased gallbladder sometimes proves effective. Useful drugs in the prophylaxis of the disturbance include quinidine, digitalis, potassium and various sedatives (Chapter XVII).

As a rule, the more rapid the tachycardia and the longer the duration of the attack, the more difficult is the management. In the active treatment of a paroxysm of tachycardia, simple procedures often prove successful and should be initiated promptly. The patient is reassured and placed in a comfortable position of rest. Measures which increase vagal tone may then be applied in the following order: carotid sinus pressure, ocular pressure, and induced vomiting. Carotid sinus pressure is by far the most effective of these methods but frequently is beneficial only after intravenous administration of a rapidly-acting digitalis preparation (cedilanid, etc.). Tachycardias at extremely high rates respond less readily to increase in vagal tone, an observation first made by Lewis.

If measures which increase vagal tone fail to end the paroxysm, sedation should be used to induce sleep Few textbooks mention the effectiveness of sedation in the treatment of tachycardia, yet we have repeatedly observed the termination of an attack following this simple measure.

When simple vagal stimulation and/or sedation prove ineffective, one of the following drugs may terminate the paroxysm. digitalis or the allied cardiac glycosides, quinidine, quinine dihydrochloride, intravenous atabrine, intranvenous morphine sulfate, intravenous magnesium sulphate, ergotamine, acetylcholine or acetyl-B-methylcholine (Mecholyl), Neosynephrine or ipecac. If the patient is in shock and absorption from oral and hypodermic routes is poor some of these drugs may be cautiously administered intravenously. The dosage and specific indications for the use of each drug are discussed in Chapter XVII.

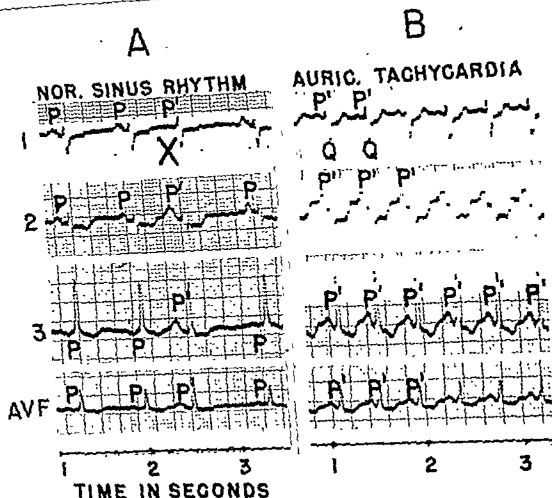


Figure 43 Electrocardiograms of patient having rheumatic heart disease associated with frequent episodes of auricular premature systoles and auricular paroxysmal tachycardia.

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auricular node. This theory was favored by Barker, Wilson and Johnston[23] in 1943 in their discussion of the mechanism of auricular paroxysmal tachycardia in man. From a consideration of "(1) the form of the auricular deflections; (2) the effects of exertion, vagal stimulation, digitalis, quinidine and other drugs upon the auricular rate and the duration of the paroxysms; (3) similarities, differences, and relations between it, auricular flutter and fibrillation; (4) the spontaneous occurrence of auriculo-ventricular block in a small number of cases and the difficulty or impossibility of producing it in most of the others; and (5) the occurrence of alternation in the auricular cycle length," they suggested that "the view that auricular paroxysmal tachycardia is caused by circus rhythm involving one of the specialized nodes – the sino-auricular or the auriculo-ventricular node – deserves serious consideration."

Hoffmann was the first to assert that in paroxysmal tachycardia, "the numerous beats were all extrasystoles and not ordinary contractions." Wenckebach[629] and Mackenzie[412] were of similar opinion.

Lewis[352, 354, 385] regarded circus movements unlikely as the basic mechanism of auricular paroxysmal tachycardia. He pointed out that the total amount of auricular muscle is not sufficient to support a circus movement in the presence of cycles the length of those which occur in auricular paroxysmal tachycardia "if a reasonable conducting rate is allowed."[385] Because of the electrocardiographic similarity between auricular premature systoles and the individual tachycardia complexes (Figure 43) and because of the frequent development of tachycardia in patients with premature beats, Lewis embraced the view that auricular paroxysmal tachycardia was simply a "run" of auricular premature systoles.

Decherd, Ruskin and Herrmann[135] plotted the momentary atrial electrical axes. Although unable to exclude the possibility of a circus path through the sino-auricular node, they felt that auricular tachycardia probably was not due to a circus movement. Scherf[542, 543] considered his observations of aconitine-produced arrhythmias incompatible with the assumption that tachycardia is caused by a circus movement.

Clearly, at present there is no general agreement among investigators concerning the nature and mechanism of auricular paroxysmal tachycardia. One possible explanation for this discord is that heretofore the course of the excitation wave in tachycardia has not been traced by means of direct experiments. In some of Lewis' experiments[353, 356] the auricle was driven electrically; in others, tachycardia followed ligation of a coronary artery. Although in the former instances post-stimulatory flutter sometimes persisted long enough for electrocardiographic study, "spontaneous" (post-stimulatory) tachycardia either failed to occur or continued for a period too short to permit adequate observations.

MATERIALS AND METHODS

In the present study the mechanism of experimentally produced auricular paroxysmal tachycardia was demonstrated by visualizing the auricle cinematographically; electrical phenomena accompanying the mechanical events were recorded on both the electrocardiograph and cathode-ray oscillograph. These observations were made during attacks of experimental tachycardia in about 40 dogs. Electrocardiograms of spontaneous auricular paroxysmal tachycardia in 107 humans were also analyzed (Chapter IX). In addition, electrocardiographic studies were made of auricular tachycardia produced experimentally in 18 patients by mechanically stroking the auricles of the exposed heart during surgical operations, the procedure and results of this undertaking are reported in detail in Chapter IV.

Auricular paroxysmal tachycardia was produced in dogs by three methods: (1) local application of aconitine; (2) electrical stimulation; and (3) mechanical stimulation.

(1) A solution of 0.05 to 0.2 per cent aconitine in benzene, on a cotton-tipped applicator, was applied externally to an arbitrarily chosen spot swabbed dry on the surface of the auricle.

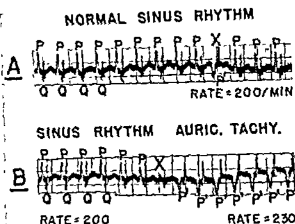


Figure 44 Lead 3

(A) Aconitine was placed on caudal portion of the dog's auricle The 10th beat (X) is an isolated auricular premature systole, P' wave is inverted The sinus P waves are all upright.

(B) Continuation of A. First 6 beats are of regular sinus origin A paroxysm of auricular tachycardia starts at X. The P' wave in the tachycardia is exactly the same as the P' wave in the isolated premature beat in A.

In several instances, the initial effect was the appearance of a few premature systoles rapidly followed by auricular paroxysmal tachycardia (Figure 44) In other cases, flutter or fibrillation developed almost immediately, by cooling or freezing the aconitine focus (Appendix), these higher rate arrhythmias were often converted into auricular tachycardia. In either event, the tachycardia usually persisted long enough to permit adequate photographic, electrocardiographic and oscillographic studies. Regardless of the site chosen as the ectopic focus, only rarely were we unable to produce tachycardia. In some experiments the aconitine was mixed with India ink to accentuate the photographic appearance of the ectopic focus.

(2) Single induction shocks from a pulse generator were sent into the wall of the auricle, at a chosen site, through a fine copper-wire electrode. As stated in Chapter II, the type of arrhythmia produced was determined by the rate of stimulation. Shock rates of less than 100 per minute* usually elicited scattered auricular premature systoles (Figure 20). Rates greater than 150 stimuli per minute usually enabled the ectopic focus to usurp the pacemaking function from the sino-auricular node, producing a paroxysm of auricular tachycardia. As a rule, the arrhythmia persisted only during the period of stimulation; in rare instances and for brief periods, it continued as a post-stimulatory effect after the shocks were discontinued.

(3) The auricles were stimulated by mechanical stroking with a wooden applicator. In all instances auricular premature beats were readily elicited. Rarely, short runs of auricular paroxysmal tachycardia were produced (Figures 29 and 45).

* Dogs anesthetized with sodium pentobarbital usually have sinus tachycardia with rates as high as 150 beats per minute The pre-anesthetic use of morphine results in slower rates, but hampers the production of the arrhythmia.

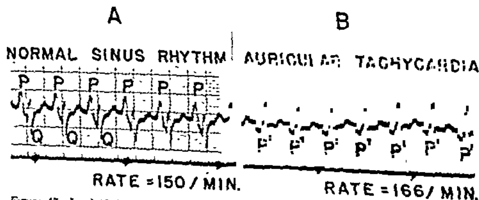


Figure 45 Lead AVF

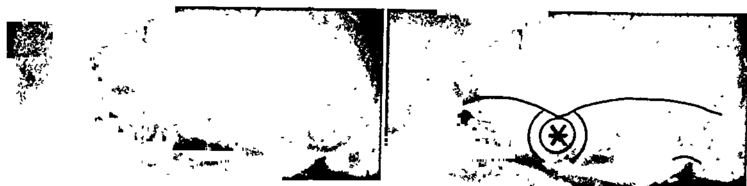
(A) Normal

(B) Auricula

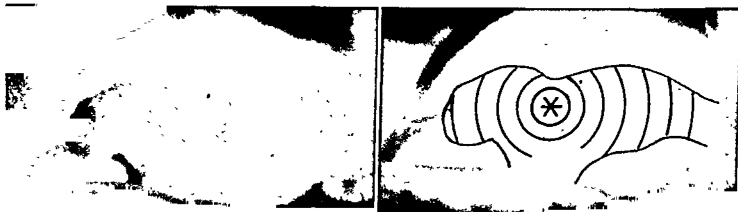
tion (stroking) c ... mechanical irrita- ... complexes exhibit



A. Diastole of right auricle.



B. Start of auricular systole The X marks the site of the application of aconitine and the onset of the contraction wave.

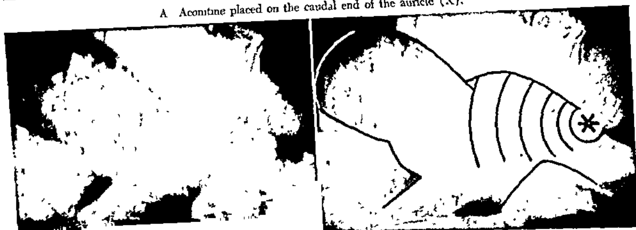


C. Auricle in complete systole. Note that the contraction is symmetrical – both sides contract at the same time

Figure 46 Auricular paroxysmal tachycardia was produced by application of aconitine at the center of right auricle (A) Diastole of right auricle (B) Auricular systole starts as a dimpling at the site of the ectopic focus (C) The contraction wave spreads concentrically in all directions and progressively involves the right auricle, it reaches the cephalic and caudal ends at approximately the same time



A Aconitine placed on the caudal end of the auricle (X).



B The contraction wave has now spread half way across the auricle shunting the blood from the caudal into the cephalic end The cephalic end is now ballooned outwards

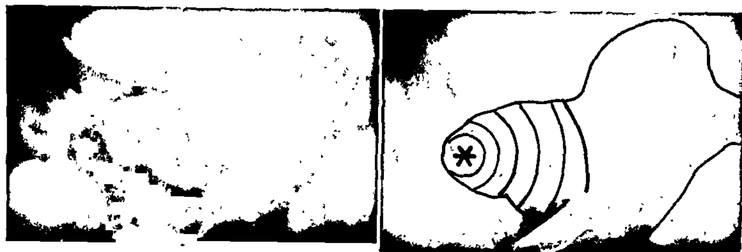


C Systole has reached the appendix which is now completely contracted, the caudal end is in diastole. It is distended with blood.

Figure 47. Auricular paroxysmal tachycardia was produced by application of aconitine at the caudal end of the auricle The tachycardia contraction wave spreads from the focus (in A) [illegible] ne auricle (in [illegible] Because the [illegible] blood is pusl[illegible] the appendix which is still in diastole (B) When the appendix contracts, the caudal end is in diastole and blood is pushed back into the caudal end (C) Because of this pooling and shuttling of blood from [illegible] tion of the [illegible]



A. The auricle is in diastole. Aconitine has been placed on the tip of the appendix, marked X.



B The contraction wave has started at point X and has now traveled half way across the auricle. Note that the caudal end of the auricle is still in diastole and that the blood from the appendix has been largely forced into the caudal end.



C. Systole has now reached the caudal end of the auricle which is contracted, forcing blood back into the appendix

Figure 48. Auricular paroxysmal tachycardia was produced by application of aconitine at the tip of the auricular appendix. Similar pooling and shuttling of blood as seen in Figure 47. In this instance, the contraction wave starts at the cephalic end of the auricle.

CINEMATOGRAPHIC APPEARANCE OF THE TACHYCARDIA CONTRACTION WAVE

The tachycardia beat begins as a wrinkling in the auricular wall in and around the ectopic focus (Figure 46). The wave of contraction spreads out from the focus in all directions simultaneously, envelops the entire auricle and, with a puckering movement, draws the chamber down toward the original focus in completion of systole. Diastole, which follows immediately, likewise begins at the ectopic focus and pursues a course identical with that of systole. The cinematographic appearance of auricular paroxysmal tachycardia from a given site is identical whether produced by aconitine or by electrical stimulation. (No films were made of the mechanically produced tachycardia.)

In this study, three main sites were chosen for the production of ectopic *foci:* (1) the center of the right auricle; (2) the caudal end of the right auricle, and (3) the right auricular appendix

(1) Center of Right Auricle: When aconitine is applied to the center of the right auricle, a dimpling appears at the site of the ectopic focus; instantly a wrinkling of the adjacent tissue occurs, spreads *in all available directions,* and progressively engulfs the entire auricle (*Figure 46*) Thus the auricular contraction wave leaves the ectopic focus, spreads concentrically in all directions, and reaches the cephalic and caudal extremities of the auricle at approximately the same time. Diastolic relaxation likewise begins at the ectopic focus and follows the same course as the systolic contraction

(2) Caudal End of Right Auricle: When aconitine is applied to the caudal end of the auricle (Figure 47), the tachycardia contraction wave from that focus spreads concentrically through the body of the auricle and finally envelops the appendix. As in the preceding experiment, diastole starts in the site of origin and follows the same course as systole.

(3) Right Auricular Appendix: when aconitine is applied to the right auricular appendix (Figure 48), the contraction wave travels in a direction diametrical to that of the wave from a focus established at the caudal end of the auricle. The appendix contracts first, the wave then spreads through the body, terminating near the inferior vena cava. Diastole follows a similar course.

In summary, the contraction wave of auricular paroxysmal tachycardia has been observed in all its details. After arising from an ectopic focus, regardless of location, the wave travels in all available directions simultaneously and not in an unidirectional circus path. In this respect the tachycardia contraction wave is similar to the waves observed in normal sinus rhythm (Chapter I) and auricular premature systole (Chapter II).

The tachycardia contraction wave differs from the normal wave in three respects: it (1) *starts* from an abnormal site of origin; * (2) repeats itself more *frequently* (higher minute rate); and (3) spreads through the auricular musculature more slowly. The tachycardia wave differs from the wave of auricular premature systole in only two respects: it exhibits (1) a higher minute rate and thereby becomes the pacemaker of the heart, and (2) a slower propagation rate.

[illegible] EARANCE [illegible]

Electrocardiograms were recorded in all instances of experimentally produced tachycardia; these included conventional limb leads and direct leads *from* the surface of the auricle. The tracings were essentially the same in pattern regardless of the method (aconitine, electric or mechanical) employed in the production of the arrhythmia, and were similar to electro-

* This statement is subject to one exception. As pointed out on page 62, auricular paroxysmal tachycardia can be elicited by placing aconitine at the [illegible] pulse [illegible] the [illegible] wave [illegible] [illegible]ations were made in a few [illegible] of auricular premature systoles (Figures 25 and 31) and auricular flutter (Figure 124).

NORMAL SINUS RHYTHM



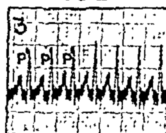


AURICULAR PAROXYSMAL TACHYCARDIA

FOCUS NEAR SINUS NODE

A





FIRST FOCUS FROZEN — SECOND FOCUS PRODUCED AT CAUDAL END OF AURICLE

B



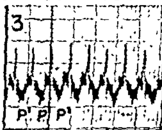


Figure 49. Auricular paroxysmal tachycardia produced by application of aconitine to two separate foci on the right auricle of the same animal.

The top photograph demonstrates the site of *impulse formation during normal sinus rhythm*. The adjacent electrocardiogram is a control tracing of normal *sinus rhythm*.

In Figure A, auricular paroxysmal tachycardia was produced *by* application of aconitine ventral to the head of the sino-auricular node, nearer the *cephalic end of the auricle* (*). *In the adjacent* electrocardiogram the P' wave is upright and in general is *of much the same shape as the control* P wave in normal sinus *rhythm*.

In Figure B, the focus on the cephalic end of the auricle was frozen with ethyl chloride spray and aconitine was *applied to the caudal end of the* auricle (*). In the adjacent electrocardiogram the P' wave is *shown to be deeply inverted*.

All electrocardiograms are from lead 3

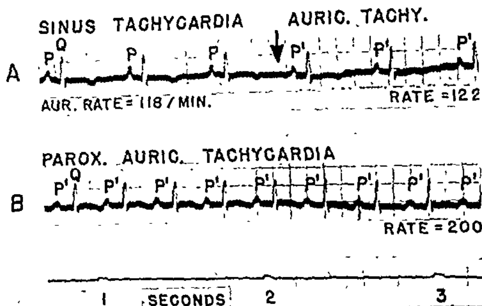


Figure 50 Lead 2 (Dog's heart).

The onset of auricular tachycardia *produced by* application of aconitine near the tail of the sino-auricular node

(A) At the 4th beat (*arrow*), the rate increases abruptly and the tachycardia begins, P' wave is almost identical *to normal* P wave.

(B) Continuation of A. The auricular rate is *increased* to 200 per minute The P' wave retains essentially the same shape but with the increase *in rate* the P'-R interval becomes prolonged

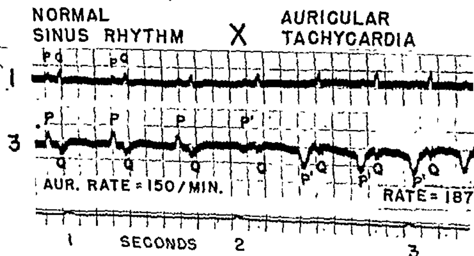


Figure 51. Leads 1 and 3, recorded ...

The onset of auricular ta...

Auricular tachycardia starts ...

... lead 3 but not in lead 1. ... inverted in

cardiograms of clinical auricular tachycardia. In 10 animals cathode-ray oscillograms were also made.

Configuration of the P' Wave in Limb Leads

In several experiments tachycardia was initiated (by the aconitine method) from two separate foci in the following manner. The first focus was created on the right auricular appendix. After the arrhythmia was produced the focus was frozen with an ethyl chloride spray; normal sinus rhythm supervened. A new focus was then produced at the caudal end of the same auricle and the *arrhythmia re-initiated* while the first focus was kept in a frozen state. The P' waves in the tachycardia from the first site were generally upright, those from the second site were inverted (Figure 49).

Thus, as in auricular premature systole, when the ectopic focus is near the site of origin of the normal impulse, the P' wave in limb leads may appear identical with the normal P waves (Figure 50). *In such instances the onset of*

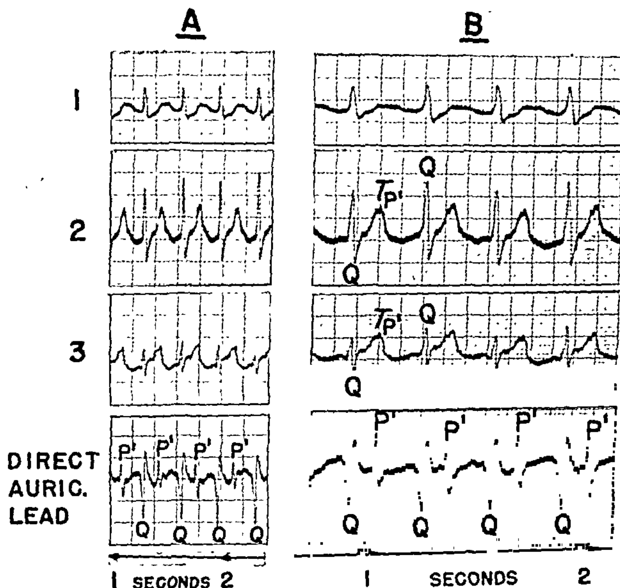


Figure 52 Electrocardiograms of auricular tachycardia (produced in a dog by the local application of aconitine) at normal (A) and increased (B) speeds

(A) Paper speed of 25 mm. per second P' wave is buried in the preceding T wave and cannot be recognized

(B) Paper speed of 50 mm per second P' wave is recognized as an irregularity in the T wave in leads 2 and 3 This is confirmed by simultaneously recorded direct auricular leads (bottom strips), the exact location of the P' waves on the T waves in leads 2 and 3 is thus clearly identified The Q-T interval is falsely prolonged

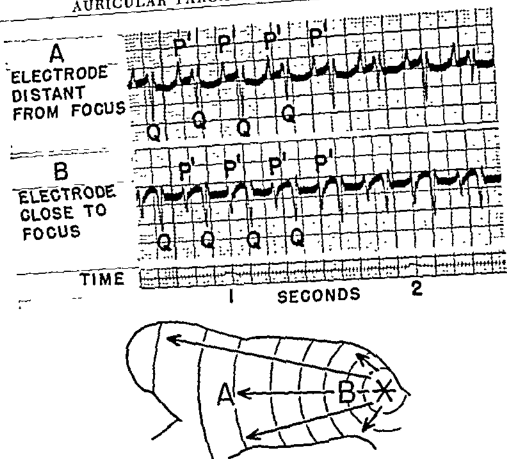


Figure 53 Demonstration of the spread of the excitation wave in auricular paroxysmal tachycardia Auricular paroxysmal tachycardia was produced by the local application of ... Two direct electrodes (A and B) were placed on ...cus, and (A) distant from the focus The intrinsic ...parently 0 03 second before that of A. The positive deflection of A is more upright than that of B These findings demonstrate that the excitation wave travels in a caudocephalic direction (from B to A). Electrocardiograms from A and B were recorded simultaneously

tachycardia is recognized solely by the sudden increase in rate Tachycardia produced elsewhere in the auricle exhibits graded variations in the shape of the P' waves When the ectopic focus is located at the extreme caudal end of the auricle the P' waves usually are deeply inverted in leads 2 and 3, in marked distinction to the normal P waves which are generally upright in these leads (Figure 51). When the ectopic focus is at the cephalic end of the auricle, the P' waves are usually upright in leads 2 and 3.

Clinical Correlation: As in auricular premature systole, the P' waves of clinical auricular paroxysmal tachycardia exhibit variations similar to those described in the experimental arrhythmia. As shown in Chapter IX, naturally occurring changes in the human are also the result of variations in the location of the ectopic focus. Unfortunately, in most tracings of clinical tachycardia recorded by the conventional electrocardiograph, the shape of the P' wave cannot be determined in the standard leads 1, 2 and 3 because the waves are superimposed on and distorted by the ventricular complexes. The use of faster paper speeds and of special auricular leads makes possible a clearer definition of the P' wave in a larger number of instances of clinical auricular paroxysmal tachycardia (Figure 52).

Configuration of P' Wave in Direct Auricular Leads

The configuration of the P' wave in direct auricular leads depends upon the distance of the electrode from the ectopic focus. Tracings from an electrode placed near the focus register primarily a negative deflection. As the electrode is moved and the distance between the ectopic focus and the electrode increases, the deflection becomes progressively more positive and the negative component eventually disappears (Figure 53). Furthermore, the time of onset of the auricular intrinsic deflection appears almost immediately after the start of the wave in leads placed near the focus; the onset is progressively later as the electrode is moved away from the focus (Figure 53). Thus, both the configuration of the P' wave and the time of onset of the intrinsic deflection in auricular tachycardia are directly related to the distance of the electrode from the ectopic focus. This general rule is of fundamental importance and is mentioned on several occasions in this monograph.

The electrocardiographic observations described above establish that the configurations of the P' waves of auricular paroxysmal tachycardia in both indirect and direct leads are similar to those of auricular premature systole (Figures 29, 30, 43 and 44); in both arrhythmias the shape of the P' wave varies with the location of the ectopic focus. The course of the excitation wave of auricular paroxysmal tachycardia as demonstrated by direct auricular leads is identical with the course of the excitation wave of auricular premature systole arising at the same ectopic focus. Clinical applications of these findings are considered in Chapter IV.

EFFICIENCY OF TACHYCARDIA CONTRACTION WAVE

Cinematographs clearly show a significant reduction in the functional efficiency of the auricle during auricular tachycardia. Several conditions contribute to this result. As discussed in Chapter II, auricular premature systole is less efficient than the normal beat because of (1) ectopic site of origin; (2) the degree of prematurity; and (3) weak or abortive contractions. In addition to these three factors, in tachycardia two other conditions prevail which adversely affect the efficiency of the beat: (1) superimposition of the auricular and ventricular beats; and (2) slower propagation speed of the contraction wave.

In many instances of auricular paroxysmal tachycardia the auricle contracts during ventricular systole, a phenomenon known as *superimposition*. An auricular contraction occurring during ventricular systole is functionally almost completely ineffective; the auricles attempt to force blood through the closed auriculo-ventricular valves which are held shut by pressures greater than the auricles can exert, so that auricular energy is expended without propelling blood into the ventricles. The cause of this phenomenon is the prolongation of the interval between auricular and ventricular contractions in a given cardiac cycle, as a result of which the auricular contraction falls just before, during, or immediately after the ventricular systole of the preceding cardiac cycle. Electrical evidence of superimposition of auricular and ventricular contractions is best seen in the oscillogram (Figures 54 and 55).

Clinical Correlation: The fact that the systole of auricular paroxysmal tachycardia is much less efficient than normal systole may have clinical significance. Under conditions of circulatory stress the normally functioning auricles may play an important part in maintaining the integrity of the circulation (Chapter I). This function is especially valuable during an emergency such as acute left ventricular failure when adequate auricular activity may support the circulation long enough to enable the ventricles to recover. Since prolonged tachycardia is apt to impose a serious burden on the heart, the coincident reduction of auricular efficiency may well aggravate the general myocardial failure occasionally seen in such arrhythmias. Post-tachycardia electrocardiographic abnormalities convincingly demonstrate the deleterious effect of this disturbance.

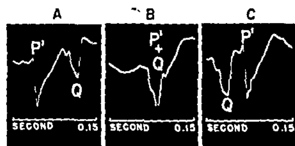


Figure 54 Cathode-ray oscillograms from a direct auricular lead in a dog with auricular paroxysmal tachycardia. The P'-Q interval is greatly prolonged so that in standard leads with ordinary equipment the record simulates nodal tachycardia The QRS initiated by an impulse from each of these P' waves would be the next succeeding one (not shown). In A the P' wave occurs just before a QRS wave In B the P' wave and QRS occur simultaneously and C the P' wave occurs just after a QRS Such fine detail is never seen with ordinary electrocardiographic equipment

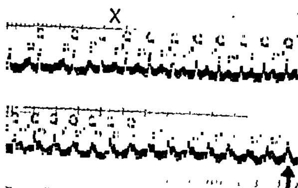


Figure 55 [illegible] strip As the tachycardia becomes more rapid the Q-Q interval becomes shorter and the P'-R interval becomes increasingly longer until in the last beat of the lower strip (marked by arrow) the P' wave is superimposed on the preceding QRS Many would interpret the lower tracing as nodal tachycardia.

OTHER TYPES OF AURICULAR PAROXYSMAL TACHYCARDIA

Nodal Tachycardia: By use of the cathode-ray oscillograph, the P'-R interval can be measured with greater accuracy than with ordinary mechanical equipment We have found that the P'-R interval is often prolonged and may vary directly with the rapidity of the tachycardia (Figure 56) This observation accounts for the frequency with which the P' wave is buried in the preceding ventricular complex. It also raises certain questions concerning the electrocardiographic diagnosis of nodal tachycardia.

According to the prevailing concept of *nodal tachycardia*, the impulse may start high, in the mid-portion, or low in the auriculo-ventricular node. If the impulse starts high, the P' wave immediately precedes the QRS; if in the mid-portion, P' and QRS occur simultaneously; if low, the P' wave follows the QRS. Since in the oscillogram the P'-R interval of auricular tachycardia often approximates or even exceeds the length of the Q-Q interval, the P' wave may occur just before, during or after the preceding ventricular complex. Thus, by use of the oscillograph it is shown that tracings of auricular tachycardia may exhibit the exact electrocardiographic patterns generally ascribed to each of three types of nodal tachycardia (Figures 54, 55 and 56). These points are given further consideration in Chapter IV.

These statements are not intended to infer that nodal tachycardia is non-existent. It is merely pointed out that the electrocardiographic configuration usually regarded as representing nodal tachycardia may also be produced by auricular tachycardia. Although true

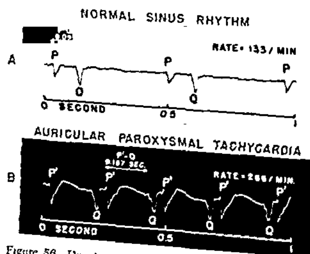


Figure 56 Direct auricular lead cathode-ray oscillograms of (A) normal sinus rhythm and (B) auricular paroxysmal tachycardia in a dog The tachycardia was produced by aconitine. In auricular paroxysmal tachycardia the P'-R interval has become so prolonged that it appears immediately after the preceding QRS and there has been a change in the shape of the P' wave Because the P' wave occurs so soon after the QRS it might be misinterpreted as low nodal tachycardia. The Q wave is of identical appearance to that in normal sinus rhythm Such details cannot be seen as clearly with ordinary electrocardiographic equipment.

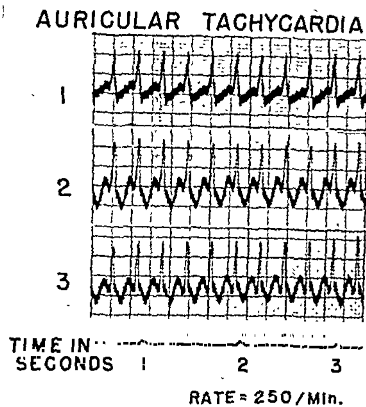


Figure 57. Auricular paroxysmal tachycardia in a dog produced by local application of aconitine. Rate 250 per minute.

Auriculo-ventricular conduction in the dog is a highly *efficient mechanism; as a result there is a ventricular response to each auricular beat even at this extremely rapid rate.*

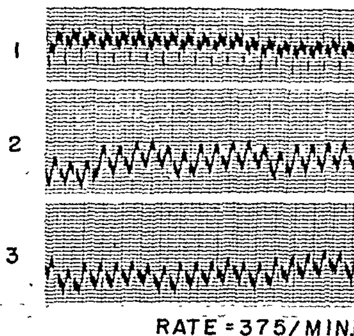


Figure 58 Auricular paroxysmal tachycardia in an infant Rate 375 per minute As in the dog, the auriculo-ventricular conduction system in infants is highly efficient This tracing is similar to that of Figure 57 The auricular deflections cannot be identified (Courtesy J J Silverman and O. M Race, and C. V. Mosby Co, publisher From *Am H J*, 37 1139, 1949)

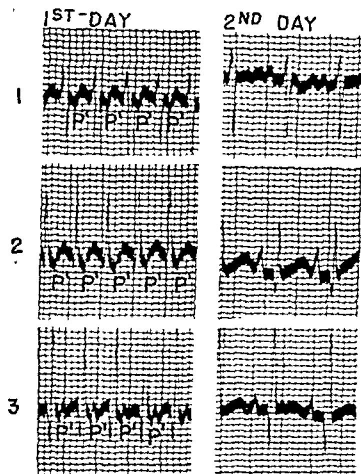


Figure 59. Auricular paroxysmal tachycardia in an infant at a rate of 300 beats per minute. EKG enlarged two times. Note that in the auricular paroxysmal tachycardia the P' waves occur shortly after the QRS This might be called nodal or auricular paroxsmal tachycardia. The record on the 3rd day shows the return to sinus rhythm (Courtesy of *Lewis T. Bullock*, M D.)

nodal tachycardia may well occur, it appears that if all parts of auricular tissue and the auriculo-ventricular node possess equal potential to become ectopic foci, on the basis of the relative area, the possibility for the arrhythmia to arise in the auriculo-ventricular *node is slight.* This much must be conceded. In the present *stage of our knowledge* it is not possible to differentiate nodal from auricular tachycardia with any degree of certainty and it is highly probable that many or most arrhythmias diagnosed as nodal tachycardia are in fact auricular tachycardia.

Tachycardia in Infants: In dogs we frequently *observe instances of rapid tachycardia* (300 or more beats per *minute)* with a ventricular response to each auricular contraction (i e., without auriculo-ventricular *block) (Fig-*

ure 57). For such an event to occur two conditions are necessary: (1) a sound auricular musculature which can contract at this rapid rate without developing fibrillation; and (2) an efficient auriculo-ventricular node, able to conduct a large number of auricular impulses per minute to the ventricles without developing auriculo-ventricular block.

A clinical manifestation of this seemingly favorable combination of conditions is the occurrence of rapid rate tachycardia in infants (Figures 58, 59, 60). Clinically, the combination of a physiologically "competent" auricular musculature and a highly efficient auriculo-ventricular node is undesirable. Failure to develop fibrillation and auriculo-ventricular block (which would reduce the ventricular rate by one-half or more) results in strain on the heart frequently sufficient to cause fatal congestive heart failure It is probable that a number of

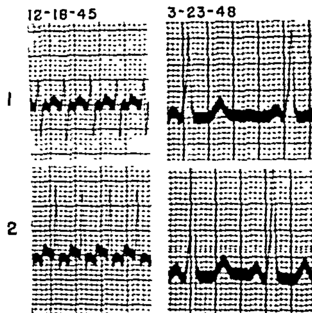


Figure 61 Auricular tachycardia in an infant at a rate of 300 beats per minute. EKG enlarged two times Twenty-seven months later the electrocardiogram reveals that the patient has the Wolff-Parkinson-White syndrome On 3-23-48, the P-R interval is 0 09 second and the QRS is 0 11 second (Courtesy of Lewis T. Bullock, M.D.)

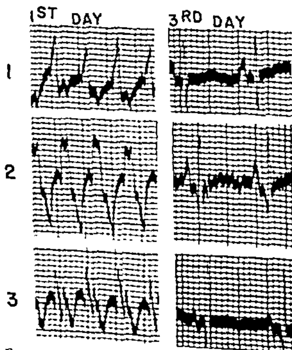


Figure 60 Tachycardia in an infant EKG enlarged two times Rate 280 per minute Because of the markedly aberrant QRS complexes this arrhythmia may be ventricular tachycardia or a supraventricular (auricular) tachycardia with aberration The differential diagnosis cannot be made with absolute certainty because of the difficulty in identifying the P' waves. The tracing on the 3rd day shows the return of normal sinus rhythm. (Courtesy of Lewis T. Bullock, M D.)

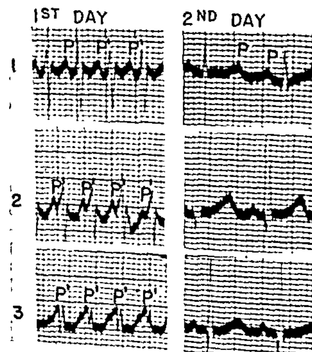


Figure 62 Wolff-Parkinson-White ... EKG ...

... might be made On the 2nd day ... Wolff-Parkinson-White syn... gram has returned to normal (Courtesy of Lewis T. Bullock, M D)

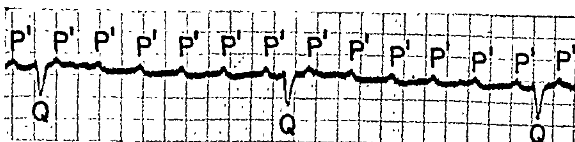


Figure 63. Auricular paroxysmal tachycardia in a patient with complete heart block following a coronary artery occlusion Auricular ectopic rhythm at a rate of 155 per minute, ventricular rate is 24 per minute.

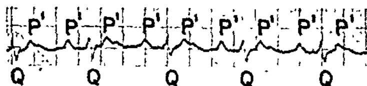


Figure 64. Electrocardiogram of a 56 year old male with arteriosclerotic heart disease. The electrocardiogram demonstrates auricular tachycardia with a partial A-V block (regular 2:1). The P' waves occur at a rate of 166 per minute, the ventricular rate is 83 per minute.

infants die of this disorder with the diagnosis unsuspected. The diagnosis in infants is often difficult because (1) their normal heart rate is relatively fast, (2) auscultation is often unsatisfactory, (3) electrocardiograms are taken infrequently, and (4) subjective complaints are not communicable Perhaps an awareness that tachycardia may be the cause of otherwise unexplained severe congestive failure in infants will aid in more frequent recognition of this disturbance.

The mechanism of tachycardia in infants is probably a congenital auricular ectopic pacemaker of inherited or inflammatory (in utero?) origin.

Tachycardia Associated with Wolff-Parkinson-White Syndrome: Auricular paroxysmal tachycardia is frequently associated with the Wolff-Parkinson-White electrocardiographic pattern *characterized by a short P-R interval* and distorted ventricular complexes (Figures 61 and 62). The ventricular complexes may retain the Wolff-Parkinson-White pattern throughout the attack of tachycardia or may become normal for the duration of the paroxysm. Tachycardia of the Wolff-Parkinson-White syndrome is often difficult to control and may even terminate fatally. At least some of the tachycardias in infants belong to this group. A full discussions of the Wolff-Parkinson-White syndrome is given in Chapter XV

Tachycardia with Heart Block: Auricular paroxysmal tachycardia may be associated with

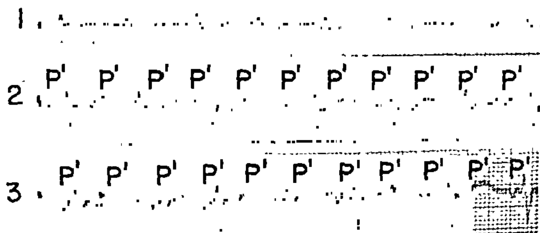


Figure 65 Auricular paroxysmal tachycardia with varying A-V block Electrocardiogram of a 75 year old male with arteriosclerotic heart disease. Electrocardiogram demonstrates a partial and varying A-V block with auricular paroxysmal tachycardia at a rate of 150 per minute.

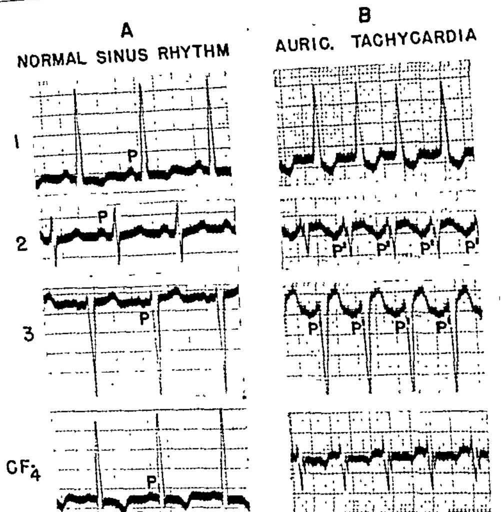


Figure 66 Demonstration of ventricular aberration occurring in auricular paroxysmal tachycardia *Electrocardiograms* of a 45 year old male with auricular tachycardia, rate 166 per minute Note that during auricular tachycardia (B), the shape of the QRS in leads 2 and CF_4 are different from those in normal sinus rhythm (A). This type of ventricular aberration is very rare in auricular paroxysmal tachycardia

auriculo-ventricular block,[132, 425] complete or partial, the latter may be regular or irregular (Figures 63, 64 and 65).

Those instances associated with partial heart block are important because of the position they occupy between ordinary tachycardia and flutter.

VENTRICULAR ABERRATION DURING AURICULAR PAROXYSMAL TACHYCARDIA

Ventricular aberration occurs occasionally in all the auricular arrhythmias, less often in tachycardia than in the others The subject is of more than theoretic interest and is discussed in detail in Chapter XV.

Two types of ventricular aberration are reflected in the electrocardiogram during auricular paroxysmal tachycardia.

(1) Ventricular aberration may begin with the onset of the tachycardia and disappear with its termination[245a] This type of aberration has not received sufficient emphasis. Its mechanism appears similar to that of the ventricular aberration accompanying *auricular* premature systole (Chapter II). It is not related to myocardial fatigue for it *disappears immediately*

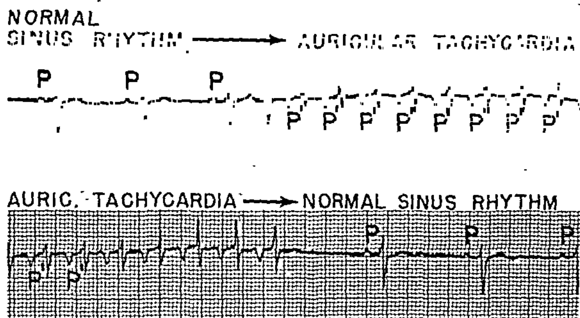


Figure 67. Continuous tracing, lead 2, showing a short paroxysm of auricular tachycardia in a child. During the paroxysm there was a marked change in the configuration of the QRS due to aberration.

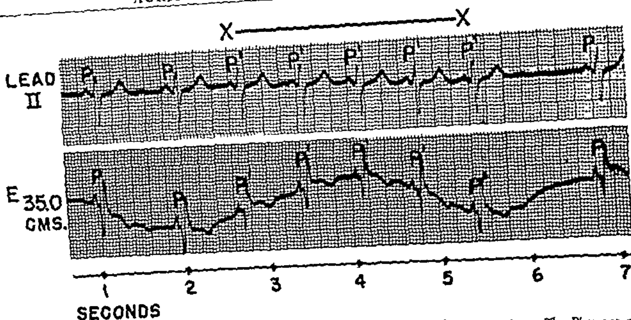
after the tachycardia reverts to normal rhythm (Figures 66 and 67). Such aberration is more common in auricular premature systoles and auricular flutter than in auricular paroxysmal tachycardia (Chapter XV). The ventricular aberration found in the Wolff-Parkinson-White syndrome may be of this type.

(2) In protracted rapid tachycardia gradual widening of the QRS complexes, with or without RS-T wave changes frequently occurs.[91, 119, 222, 574] The most common changes are depression of the RS-T segment with flattened or inverted T waves in the standard limb leads and usually in leads AVL and AVF. The RS-T segment and T wave may be elevated in lead AVR. These changes may persist for some time after the arrhythmia has been terminated (post-tachycardia electrocardiographic syndrome). This type of ventricular aberration occurs most frequently in diseased hearts and is believed to be due to myocardial fatigue (Figure 42).

DISCUSSION

Both cinematographically and electrocardiographically, the tachycardia wave is identical with the wave of the premature systole These observations, together with the fact that both arrhythmias can be produced by the same methods, demonstrate the existence of a definite relationship between the two disturbances. This is consistent with the common clinical experience that auricular premature systoles tend to precede or follow paroxysms of auricular tachycardia. Frequently, paroxysms are of such short duration that they are overlooked by both patient and physician, but are fortuitously revealed in the electrocardiogram. Such short paroxysms usually are of relatively slow rates, only slightly faster than normal sinus rhythm. Not uncommonly, a single tracing will exhibit scattered premature beats in one part and "runs" of tachycardia in another. Similar evidence led Lewis to regard auricular tachycardia as a "run" of premature systoles; our observations confirm this conclusion.

Clinically, it has long been known that both the onset and termination of auricular paroxysmal tachycardia are sudden. Our experimental observations suggest the following explanation for the abruptness of these changes It has been shown that experimental auricular premature systole and auricular paroxysmal tachycardia have essentially the same mechanism and that in any given instance the emergence of either arrhythmia depends upon the *relative rates of discharge from the normal and ectopic pace-*



at a slow rate of 90 beats per minute. The P' waves of the tachycardia are of slightly different shape than the P waves of the normal sinus rhythm preceding the paroxysm, suggesting that the auricular tachycardia arose from an ectopic focus close to the sinus node.

makers. By analogy, in a representative clinical case, when the sinus rate is approximately 80 beats per minute and an ectopic focus which discharges at a rate of 50 stimuli per minute develops, the great majority of impulses from the ectopic focus will be ineffective because of the refractory state of the auricular musculature. Only occasional, scattered, premature systoles will result. When the rate of discharge from the ectopic focus rises *suddenly* to a level *greater* than that from the sino-auricular node, the ectopic focus abruptly usurps the pacemaking function and the arrhythmia becomes dominant. The normal sinus impulses are rendered ineffective by the refractory periods due to the rhythm from the ectopic focus, and auricular tachycardia prevails. When the rate of discharge of the ectopic focus falls below that of the sinus node, the termination of the arrhythmia occurs abruptly. The above sequence of events was repeatedly reproduced in animal experiments by means of electrical stimulation in which the rate of discharged stimuli could be controlled at will (Figure 20) Similar observations were made during the course of aconitine-produced arrhythmias in dogs, abrupt changes from one arrhythmia to another occurred either spontaneously or after the aconitine focus was cooled. Undoubtedly, in the aconitine experiments also, the underlying factors were sudden variations in rate of stimulation occurring either spontaneously or as the result of the temperature changes. From these observations it is clear the auricular paroxysmal tachycardia can occur at rates only slightly more rapid than normal sinus rhythm. This has been reported previously,[452] and we have seen short bursts of tachycardia in a patient with rates as low as 90 beats per minute (Figure 68). In these instances the tachycardia is of short duration, lasting only a few beats, and is usually detectable only by the electrocardiogram. The faster the rate, the longer is the duration of the paroxysm and the more unfavorable the prognosis. An understanding of the basic mechanism explains this phenomenon. In some institutions, tachycardia is diagnosed only if the rate exceeds a definite limit, such as 150 beats per minute. As demonstrated here, such rules are obviously false. It is now possible to define auricular paroxysmal tachycardia as an arrhythmia accompanied usually by a 1:1 ventricular response

and arising from an ectopic focus which is discharging at a regular rate greater than the sinus rate.

SUMMARY AND CONCLUSION

Auricular paroxysmal tachycardia has been produced in dogs by three methods: (1) application of aconitine; (2) electrical stimulation; and (3) mechanical stimulation; it has been studied by means of the high-speed motion picture camera, the electrocardiograph and the cathode-ray oscillograph. Whether produced by aconitine or by electrical stimulation, the arrhythmias are visually and electrocardiographically identical. The wave starts from an ectopic focus which can be experimentally produced at will on any part of the auricle. *From the ectopic focus the wave spreads in all available directions simultaneously until the entire auricle is involved.* As demonstrated in electrocardiograms from direct auricular leads, the electrical wave is similar in origin and course to the mechanical wave arising at the same focus. The general character and appearance of both the contraction wave and the excitation wave vary with the site of origin. Since the wave travels in all available directions, when it originates at one end of the auricle it sweeps toward the opposite end, when the site of origin is in the center of the auricle the wave moves in all directions toward the periphery. Thus, *the wave of auricular paroxysmal tachycardia does not pursue a unidirectional path, and therefore cannot be the result of a circus movement.*

The tachycardia contraction wave and the normal sinus wave are similar in that both spread in all available directions simultaneously; they differ in that the tachycardia wave arises in an ectopic focus, repeats itself more frequently, and has a slower rate of propagation. To the extent that the tachycardia wave differs from the normal in these respects, its efficiency is impaired. This impaired efficiency may play a significant role in aggravating the general myocardial failure which occasionally occurs in prolonged clinical tachycardia.

The contraction wave of tachycardia bears a closer resemblance to that of auricular premature systole than to that of normal sinus rhythm in that the waves of both tachycardia and premature systole arise from an ectopic focus; the waves of the two arrhythmias differ in that in tachycardia (1) the rate of discharge is more rapid and the ectopic focus becomes the pacemaker of the heart; and (2) the speed of propagation is slower. The transition from premature systoles to tachycardia is directly related to the rate at which the impulses are discharged from the ectopic focus. The former disturbance occurs when the rate of the abnormal rhythm is less than that of the sinus rhythm, the latter when the rate of the arrhythmia exceeds that of the sinus rhythm. Changes in these rates may account for the abruptness which characterizes the onset and termination of clinical auricular tachycardia.

By means of the cathode-ray oscillograph it was found that auricular tachycardia with prolonged P'-R interval can simulate nodal tachycardia. Many instances regarded as nodal tachycardia may in fact be auricular tachycardia.

CHAPTER IV

Experimental Production of Auricular Premature Systoles and Auricular Paroxysmal Tachycardia in Man

The experimental production of a deranged function in animals greatly facilitates understanding of the corresponding disturbance in man However, a shadow of doubt always remains concerning the comparability of a disturbance produced in the animal with that found in man. When the disorder can be produced in an innocuous fashion in human subjects, and if it is identical with the spontaneously occurring disturbance, the significance of the experimental [illegible] human dis[illegible] ately, few [illegible] cular diseases or disturbances can be studied *in man* because of the deleterious effects on the subject. [illegible]

A thorough study of experimentally produced arrhythmias in man has never been done Auri[illegible] ture contains no reports of experimental auricular paroxysmal tachycardia *in man*

In the present study, auricular premature systole and auricular paroxysmal tachycardia were produced by mechanical stimulation of the auricles of 18 patients undergoing operation on the heart or lungs The pericardium was opened in 13 instances The remaining five patients had tuberculosis, here, the pericardium was not opened because of danger of extension of the infection In these five patients the various parts of the auricles could be identified readily and the arrhythmias produced without difficulty through the semi-transparent parietal pericardium In all 18 patients the auricles were stimulated at three places—the appendix, the center of the body, and the caudal end – by gentle irritation with the tip of a blunt forceps or by manual pinching. The entire experiment generally lasted no longer than two to five minutes. In every instance the auricles responded readily to this innocuous procedure and the two arrhythmias were produced from the selected sites at will.

Simultaneous leads 1, 2, 3 and AVF were recorded during all experiments and esophageal lead electrocardiograms were taken in 10 instances. In addition, high-speed cinematographs of the exposed auricles were recorded in two patients with experimentally produced auricular premature systole. In all 18 patients the results were essentially similar, with only minor variations.

As described in Chapter II, the high-speed cinematographs of experimentally produced auricular premature systole in man, like those in the dog, showed that the contraction wave arose at the site of stimulation and traveled outward in all available directions (Figures 21, 22, 24, and 35). The results of our electrocardiographic study of experimental auricular premature systole and auricular paroxysmal tachycardia in man are discussed in the present chapter.

THE LIMB LEAD [illegible] OF [illegible] IN MAN

The premature systole is the simplest auricular arrhythmia; knowledge of its mechanism provides the foundation for an understanding

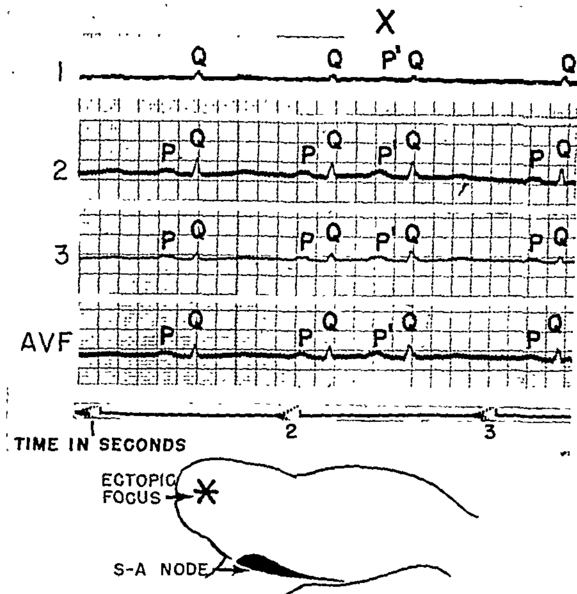


Figure 69. Premature systole in man produced by stimulating the right auricular appendix The 3rd beat (X) is a premature systole. The P' wave is of almost identical configuration to the normal P wave. The P'-R interval is prolonged.

of the more complicated and serious arrhythmias. Consequently, it is of prime clinical importance to determine the location of the ectopic foci of spontaneous auricular premature systoles by correlating the configuration of their P' wave with the P' waves of experimentally produced arrhythmias with known sites of the ectopic focus.

In the study of ventricular premature beats, the direct application to man of the results of experiments in the dog led to erroneous interpretation of electrocardiograms for many years.[320] In order to avoid a similar error with respect to the auricles, and since the auricular arrhythmias are more common than the ventricular arrhythmias, we deemed it essential to examine thoroughly and clarify the mechanism of the disturbance in man. This study was accomplished without difficulty in the manner previously described.

Observation 1: Configuration of the P' Wave in Limb Leads

Premature Systoles from the Appendix of the Right Auricle: Premature systoles produced in this region exhibited upright P' waves in leads 1, 2, 3 and AVF (Figure 69). The configuration of these waves resembles that recorded during

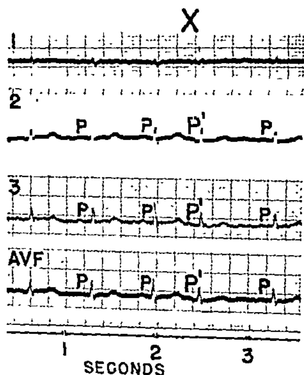




Figure 70 Premature systole in man produced by stimulating the tip of the left appendix The 4th beat (X) is a premature systole The P' wave is similar to the normal P wave The P' wave is less upright than the normal ones shown

normal sinus rhythm The P' waves of the premature beat are distinguishable because of (1) their prematurity, and (2) slight variations in contour As a rule, the P'-R intervals are longer than those of the normal sinus beats

Focus on the Left Appendix: Premature beats from the left appendix, like those from the right appendix, are characterized generally by upright P' waves in leads 1, 2, 3 and AVF (Figure 70). The configuration of the P' wave is similar to that of the normal P wave and the P' waves of premature systoles produced in the right auricular appendix (Figure 69). The P'-R interval is generally prolonged.

Focus on the Mid-Portion of the Right Auricle: The center of the right auricle was next stimulated. This area was generally 1 or 2 centimeters ventral to the sinus node. In most cases the premature systoles from this point were *isoelectric in* all leads and P' waves could not be identified with certainty. If deflections were produced at all, they were barely perceptible positive or negative waves. In these instances, since the P' wave cannot be identified, the P'-R interval cannot be measured (Figure 71).

Focus on the Mid-Portion of the Left Auricle: Premature systoles from the mid-portion of the left auricle are similar to those from the analogous part of the right auricle, i.e., the P' wave

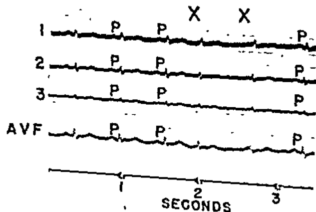




Figure 71 Premature systoles in man produced by stimulating the body of the right auricle The 4th and 5th beats (X) are premature systoles The P' waves are almost invisible The premature beats are distinguishable from the normal beats by (1) the absence of an auricular deflection, and (2) the degree of prematurity of the ventricular complex It is impossible to measure the P'-R interval

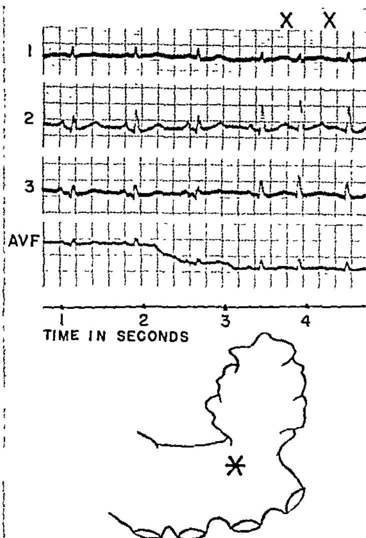


Figure 72 Premature systole in man produced by stimulating the center of the body of the left auricle The 5th and 6th beats (X) are premature systoles. The P′ wave cannot be identified with certainty. Ventricular aberration is present

is isoelectric or almost so in the limb leads. The P′ waves cannot be clearly seen and as a result the P′-R interval cannot be measured (Figure 72)

Focus on the Caudal Portion of the Right Auricle: Stimulation of the caudal portion of the right auricle consistently produced inverted P′ waves in leads 2, 3 and AVF (Figures 73, 74 and 75); the waves in lead 1 usually were isoelectric. The deflection was generally in a direction opposite that of the waves of normal sinus rhythm. The P′-R intervals here as a rule were shorter than those of sinus beats, often as short as 0.08 second (Figures 74 and 75).

Focus on the Caudal Portion of the Left Auricle: Premature systoles produced by stimulation of the caudal portion of the left auricle exhibited inverted P′ waves in leads 2, 3 and AVF with P′-R intervals which were usually 0.08 to 0.12 second in duration (Figure 76). The configuration of these waves is the same as that obtained when the caudal part of the right auricle was stimulated (Figures 73, 74 and 75).

The above observation establishes that in human subjects in the limb leads the *P′ waves of premature systoles from analogous portions of the right and left auricles have similar configurations.* Waves from the cephalic region are upright, in fact, more upright than the normal P wave; *those from the center are biphasic*

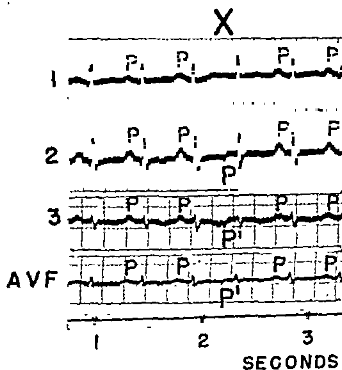


Figure 73 Premature systole in man produced by stimulating the caudal end of the right auricle The 4th beat (X) is a premature systole. *The P′ wave is inverted in leads 2, 3 and AVF and invisible in lead 1*

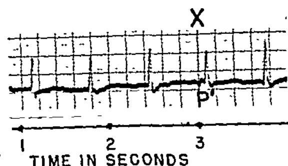




Figure 74 Premature systole in man produced by stimulating the caudal end of the right auricle, Lead 3 The 4th beat (X) is the premature systole The P'-R interval is as short as 0 08 second Beats of this nature are frequently diagnosed as high nodal premature systole or coronary sinus rhythm, but are actually auricular premature systoles from the caudal end of the auricle

or isoelectric; those from the caudal end are deeply inverted. A comparison of this observation with observation 1 in Chapter II indicates that the auricles in the dog are electrocardiographically similar to those in man. This contrasts with the marked electrocardiographic difference between the ventricles of man and dog.[339]

Observation 2: The Electrical Axes of the Human Auricle During Experimentally Produced Auricular Premature Systole

The total electrical axes of the auricles during normal sinus rhythm and during an experimentally produced premature systole were easily calculated from simultaneous leads 1, 2 and 3. For this purpose the electrocardiograms were magnified (2 times) and the Einthoven triangle was utilized in a routine manner.

Focus at the Cephalic End: In each instance in which the ectopic focus was at or near the sinus node or in either appendix, the P' waves

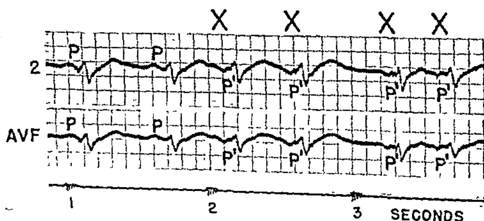




Figure 75 Premature systoles in man produced by stimulating the caudal end of the right auricle The 3rd, 4th, 5th and 6th beats (X) are premature systoles. The P'-R interval is as short as 0 08 second. The P' waves are inverted [illegible]

nature

Actually

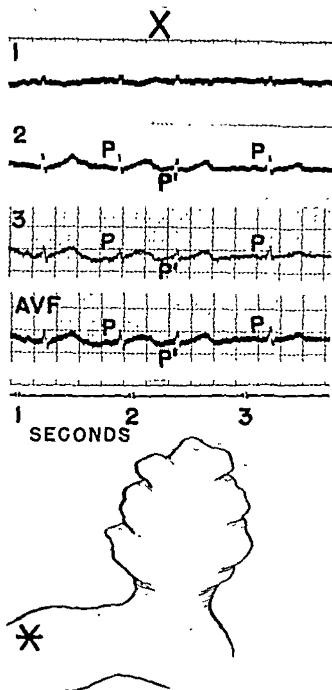


Figure 76. Premature systole in man produced by stimulating the caudal end of the left auricle. The 3rd beat (X) is the premature systole. The P' waves are inverted in leads 2, 3 and AVF

of the premature systoles resembled the normal P wave and were upright in leads 1, 2 and 3. According to current concepts of electrocardiography, therefore, the impulse must have traveled along a cephalocaudal direction. The electrical axis of the premature beat starting in this region is similar to that of the normal sinus beat (Figure 77).

Focus at the Center of Each Auricle: When the ectopic focus is midway between the extremities of the auricle, the impulse travels in all directions — *caudal, cephalic, and to either side* — with approximately equal potentials. The potentials tend to nullify each other on the electrocardiogram, with the result that little or no P' wave is inscribed. Instead the record shows *only an isoelectric line or a minute deflection.* Figure 78 shows this phenomenon calculated from the classic Einthoven Triangle.

Focus at the Caudal End: In those instances in which the ectopic focus was at the caudal end of either auricle, the P' waves were inverted in leads 1, 2, and 3. Hence, the impulse must have traveled *in a caudocephalic direction.* Calculation of the total atrial electrical axis shows the direction of the impulse to be almost opposite that of the impulse of normal sinus rhythm (Figure 79).

The P'-R Interval: The P'-R interval may be prolonged in some instances (Figure 69). In general, the more caudal the ectopic focus, the shorter is the P'-R interval. This phenomenon may be due in part to the proximity of the caudal portion of the auricle to the auriculoventricular node (Figures 74 and 75).

Observation 3: Ventricular Aberration in Auricular Premature Systoles in Man

Examination of the records of experimentally produced auricular premature systole in man *shows that ventricular aberration* occurs in some instances (Figure 72) and not in others. This is true also of the spontaneously occurring disturbance in man. More thorough discussion of this subject is presented in Chapter XV.

Observation 4: Auricular Premature Systole Simulating Nodal Premature Beats

At the present time it is postulated that premature beats may start in the auriculo-ventricular node itself.[98, 127] *If the P' wave occurs* immediately (less than 0.12 second) before the QRS complex the ectopic focus is thought to be high in the node. If the P' waves occur during the QRS, the beat is believed to originate in

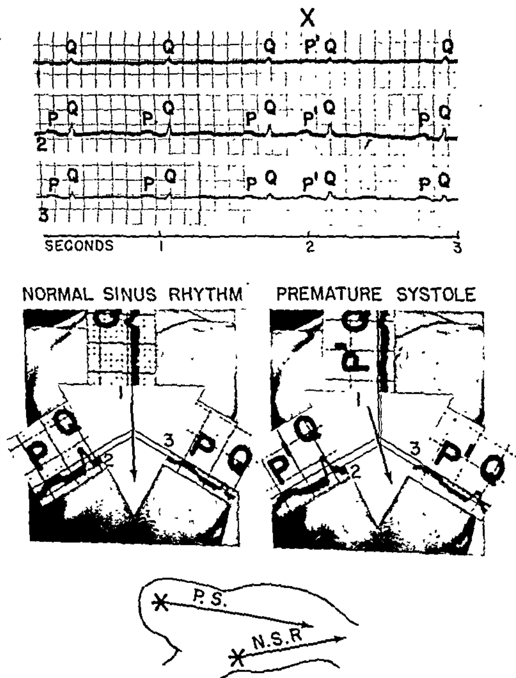


Figure 77 Premature auricular systole in man produced by stimulating the right appendix. The premature systole is the 4th beat (X); the P' wave is upright in leads 1, 2 and 3. A comparison of the total atrial axes of normal sinus rhythm and premature systole as calculated from *the classic Einthoven triangle* is shown below. It can be seen that the impulse of both *rhythms is in a cephalocaudal direction*. The electrocardiograms superimposed on the picture of the chest are enlarged two times. The *lengths of the* arrow in the triangles have been magnified for clarity and show direction only.

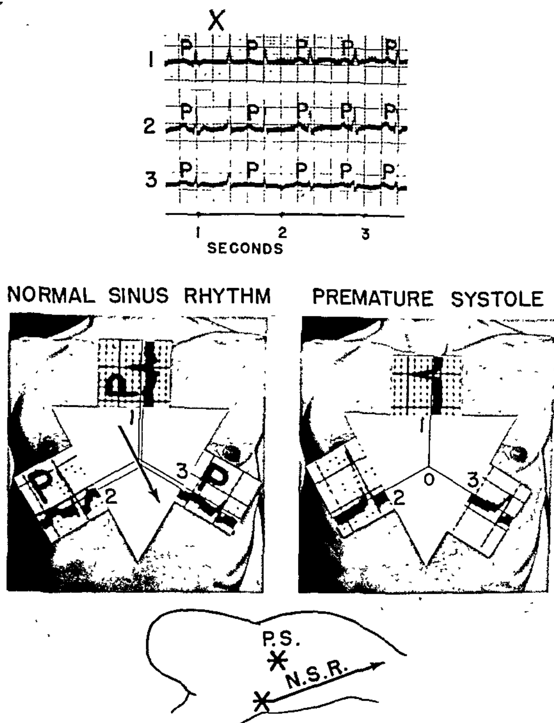
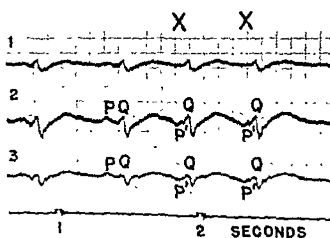
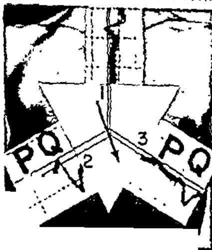


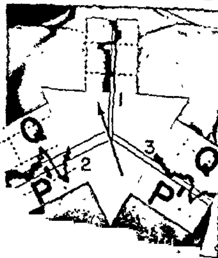
Figure 78 Premature systole in man produced by stimulating the center of the right auricle The second beat (X) is the premature systole The P′ waves are not visible Ventricular aberration is present. The total atrial axes are calculated below for normal sinus rhythm and for the premature systole Since the P′ waves of the premature systole are invisible, the atrial axis is zero Thus, with the ectopic focus located in this region, the P′ wave is either invisible or of very low magnitude



NORMAL SINUS RHYTHM



PREMATURE SYSTOLE



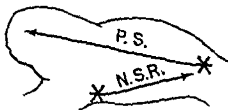


Figure 79 ...
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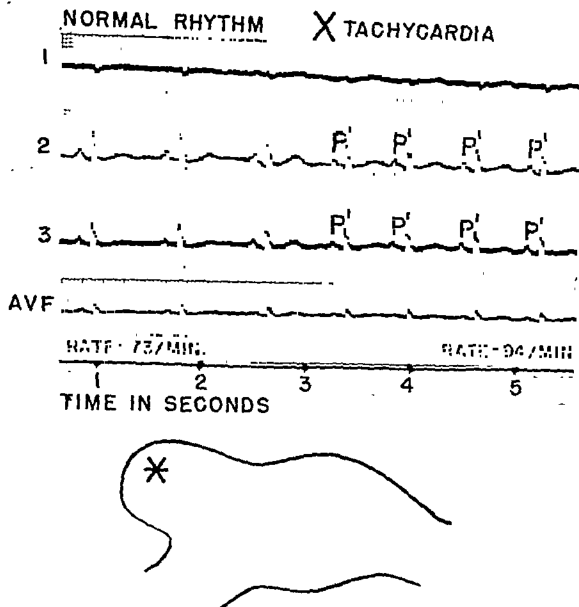


Figure 80 Auricular paroxysmal tachycardia in man produced by stimulating the right auricular appendix The normal sinus rhythm rate is 73 beats per minute The onset of the tachycardia is at the 4th beat, the rate is 94 beats per minute The P' wave is upright in leads 2, 3 and AVF, the P'-R interval is 0 14 second

the middle of the auriculo-ventricular node. If the P' waves immediately follow the QRS, the beats are supposed to arise low in the node. We have repeatedly produced the electrocardiographic configuration ascribed to high nodal and mid-nodal premature beats while stimulating the auricular wall remote from the auriculo-ventricular node.

As shown in Figures 71 and 72, premature systoles starting in the middle of the right and left auricles are generally isoelectric, and as a result the P' waves are not perceptible. The electrocardiogram here is identical with that classically described as characteristic of mid-nodal premature beats. Similarly, the picture of high nodal premature systoles with inverted P' waves and short P'-R intervals has been produced repeatedly by stimulating the caudal end of either auricle In these instances the P' wave is deeply inverted and the P'-R interval may be less than 0 12 second. Figures 74 and 75 show two such examples with P'-R intervals of 0 08 second

Thus the electrocardiographic configuration of high or mid-nodal premature systoles is duplicated by premature systoles arising in the

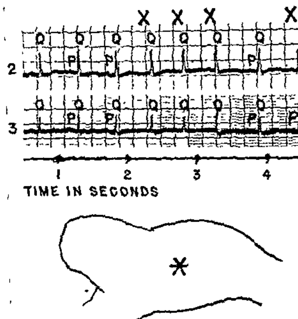


Figure 81 Auricular paroxysmal tachycardia in man produced by stimulating the middle of the right appendix The tachycardia is of three beats duration (X) P′ waves are not clearly visible, the onset and offset of tachycardia are distinguishable by the sudden changes in rate

auricle proper Furthermore, it is possible that some electrocardiograms interpreted as showing low nodal premature beats may actually reflect auricular premature beats in which the P′-R intervals are long and the P′ waves immediately follow the preceding QRS complex (Figures 54, 55 and 56)

OBSERVATION 5 AURICULAR PREMATURE SYSTOLE SIMULATING CORONARY SINUS RHYTHM

The similarity between the complexes produced when the ectopic focus is at the caudal end of the auricle and those of "coronary sinus rhythm" is worthy of note. It will be recalled that in coronary sinus rhythm the P′ waves are inverted in leads 2 and 3, and are isoelectric or low-upright in lead 1.[340] We have observed this type of configuration when the impulse arises at the caudal portion of either the right or the left auricle. Therefore, the electrocardiographic appearance of coronary sinus rhythm is found not only when the coronary sinus itself is the site of impulse formation, but also when areas adjacent to it in the caudal region of both auricles are irritated and become ectopic foci. Thus it would seem that there is no specific entity of coronary sinus rhythm.

THE L[illegible]
OF [illegible]
[illegible]
[illegible]

Paroxysms of auricular tachycardia were produced on 12 occasions in 8 subjects by stimulating various parts of the auricles during thoracic surgical procedures. One cannot predict in advance of stimulating the auricle whether isolated premature beats or short runs of tachycardia or both will occur, either rhythm is produced with ease. In 11 of the 12 instances the paroxysms of tachycardia were of short duration, lasting only a few beats; in the remaining instance the paroxysm persisted approximately two hours. In no case was the patient harmed as a result of the procedure.

OBSERVATION 6. CONFIGURATION OF THE P′ WAVE OF EXPERIMENTALLY PRODUCED AURICULAR PAROXYSMAL TACHYCARDIA IN MAN

Focus on the Right Auricular Appendix: Figure 80 shows a paroxysm of auricular tachycardia produced by stimulating the appendix of the right auricle in a patient. The P′ waves of tachycardia from this location are upright in leads 2, 3 and AVF, often more upright than the normal P wave. Thus the P′ wave of auricular tachycardia from the right auricular appendix is similar to that of a premature systole produced in the same region.

Focus on the Center of the Auricles: Auricular paroxysmal tachycardia was produced by stimulating the centers of both the right and left auricles. Paroxysmal tachycardias from this location in either auricle are electrocardiographically similar; the P′ waves are isoelectric, (Figure 81) or show only slight undulations in the base line (Figure 82), or are inverted (Figure 83B).

Focus on the Caudal End of the Auricles: Auricular tachycardia was produced by stimulating the caudal region of either the left or the right auricle on at least six occasions. The P′ waves of tachycardia from this region are

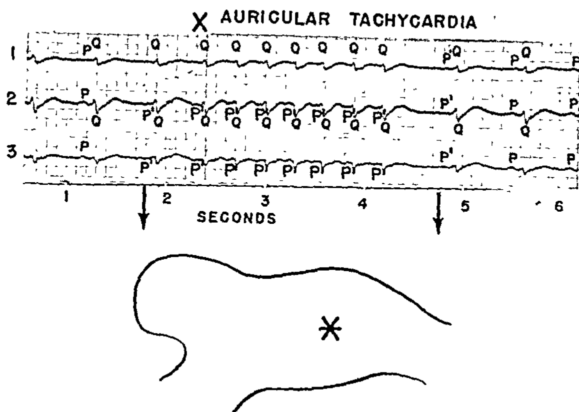


Figure 82. Premature systoles and a short burst of tachycardia in man produced by stimulating the center of the right auricle (illustration reduced to half size). The normal P wave (2nd beat) is upright The first premature systole (first downward arrow) is the third beat. The run of tachycardia begins at X. The first beat after the tachycardia (second arrow) is also a premature systole The P' waves during tachycardia and during premature systole are barely visible and are slightly inverted. The focus in this case was probably from the center of the auricle.

deeply inverted in leads 2, 3 and AVF; the P'-R interval is generally short, often shorter than normal.

In Figure 84A, showing sinus tachycardia, the auricular rate is 115 beats per minute, the P wave is upright in leads 2, 3 and AVF, and the P-R interval is 0.20 second. In Figure 84B, recorded from the same patient after tachycardia was produced by stimulation of the caudal region of the auricle, the auricular rate is 136 beats per minute, the P' wave is inverted in leads 2, 3 and AVF, and the P'-R interval is reduced to 0.10 second. Figure 85 shows another example of tachycardia produced in the caudal region; again the deeply inverted P' waves and short P'-R interval are apparent. In Figure 85 the second beat (X) is a premature systole with an inverted P' wave in leads 2, 3 and AVF. Starting at the fourth beat is a paroxysm of auricular tachycardia lasting three beats. The P' waves of the tachycardia are identical with those of the premature systole. The P'-R interval is 0.08 second in both the auricular premature systole and the paroxysm of auricular tachycardia.

From the above observation it is apparent that in a given subject the P' waves of auricular premature systoles and paroxysmal tachycardia from the same ectopic focus are identical. This was repeatedly seen in those instances in which premature beats alternated with short paroxysms of tachycardia (Figures 82 and 85). The same observation has been made consistently in the dog (Figures 29 and 30).

The atrial electrical axes in tachycardia and in auricular premature systoles are identical also because the P' waves are identical. Whatever the nature of the electrical activity in one disturbance, it is the same in the other. Tachycardia is simply a "run" of premature systoles occurring when the ectopic focus discharges more rapidly than the sinus node and

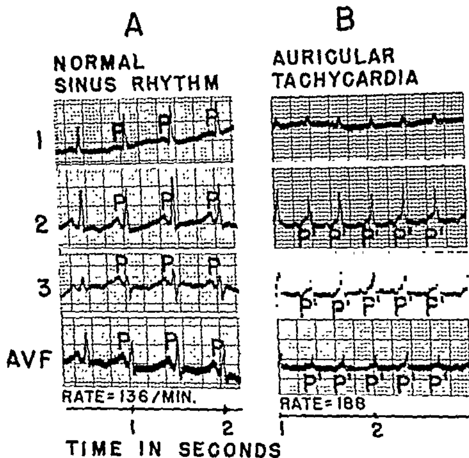


Figure 83. Electrocardiograms from a patient during thoracic surgery.

(A) Normal sinus rhythm. The P wave is upright in leads 1, 2, 3 and AVF.

(B) Auricular paroxysmal tachycardia in man produced by stimulating the center of the right auricle. The P' wave is biphasic or inverted in leads 2, 3 and AVF and isoelectric in lead 1. The P'-R interval is somewhat reduced. Ventricular aberration is present, most marked in leads 1, 3 and AVF.

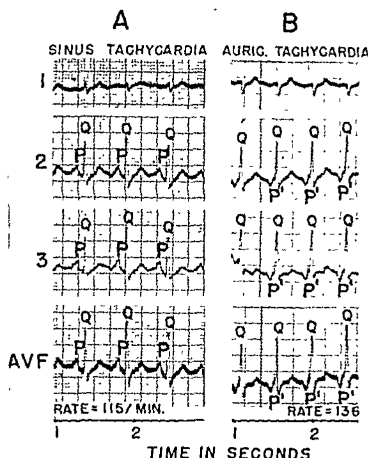




Figure 84 Electrocardiograms from a patient during thoracic surgery

(A) Sinus tachycardia The P wave is upright in leads 2, 3 and AVF The P-R interval is 0 20 second

(B) Auricular paroxysmal tachycardia in the same patient, produced by stimulating the caudal end of the right auricle The P′ wave is deeply inverted in leads 2, 3 and AVF, the P′-R interval has been reduced to 0 10 second

therefore takes over the pacemaking function. The individual beats in the two disturbances are identical.

Observation 7: Auricular Tachycardia Simulating Nodal Tachycardia

As with nodal premature beats, it is postulated that nodal tachycardia may be diagnosed and localized according to the configuration and timing of the P′ wave. If the P′ wave appears just before the QRS complex and the P′-R interval is shorter than normal, high nodal tachycardia is said to be present. In so-called mid-nodal tachycardia, the P′ wave is buried in the QRS complex. When the P′ wave is inverted and immediately follows the QRS complex, low nodal tachycardia is diagnosed. As in our experiments with premature beats, auricular tachycardias produced by stimulation at the mid-portion and caudal end of the auricles were electrocardiographically similar to mid-nodal and high nodal tachycardia, respectively. The P′ waves cannot be clearly seen in tachycardias from an ectopic focus in the middle of the auricle (Figures 81, 82 and 83). In such electrocardiograms, since the P′ wave is not perceptible, mid-nodal tachycardia might be erroneously diagnosed. In tachycardia resulting from stimulation of the caudal region of either auricle, the P′ waves are inverted in leads 2, 3 and AVF, and the P′-R interval is usually shorter than normal (Figures 84 and 85). The electrocardiographic record here is identical with that considered characteristic of high nodal tachycardia.

This observation discloses that many or most arrhythmias now diagnosed as high or mid-nodal tachycardia are, in fact, some type of auricular tachycardia. It is also significant that auricular tachycardia starting at the caudal end of the auricle can simulate what is now called coronary sinus tachycardia.

THE ESOPHAGEAL LEAD ELECTROCARDIOGRAM OF EXPERI- ... PRE- ... AR

In 10 human subjects, esophageal leads from high, low and mid-auricular levels were recorded simultaneously with either lead 3 or lead AVF during the production of auricular premature systole or auricular paroxysmal tachycardia. The esophageal electrodes and techniques employed are described in the Appendix.

In order to explain the significance of the esophageal lead electrocardiograms reproduced

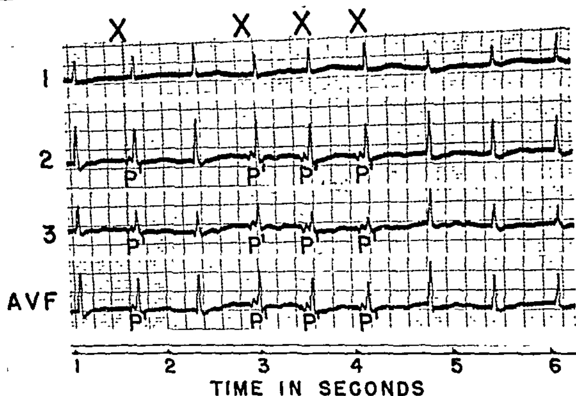




Figure 85 Auricular paroxysmal tachycardia from the caudal end of the right auricle The second beat is a premature systole, auricular tachycardia of three beats duration is present from the 4th to the 6th beats The P' wave is identical in premature systole and in auricular tachycardia, the P'-R interval is reduced to 0 08 second Ventricular aberration is present

in this chapter, the shape of the normal P wave in such tracings must first be described (Figure 86).[471] Esophageal lead tracings of normal sinus rhythm recorded from the highest auricular levels exhibit a deep negative P wave (Figure 86), this may or may not be preceded by a small positive deflection Tracings from mid-auricular levels are characterized by a diphasic P wave. In tracings from the lowest auricular levels the positive wave is large while the negative deflection is small or absent. Esophageal lead electrocardiograms recorded from levels cephalad to the auricles show a simple monophasic negative P wave, tracings recorded caudad to the auricles display a simple monophasic upright P wave. The form of these normal deflections is understandable since the *sino-auricular* node is located just antero-laterally to the esophagus, and the main course of the electrical wave of normal *sinus rhythm is in* a cephalocaudal direction. Thus the normal wave travels away from an esophageal electrode

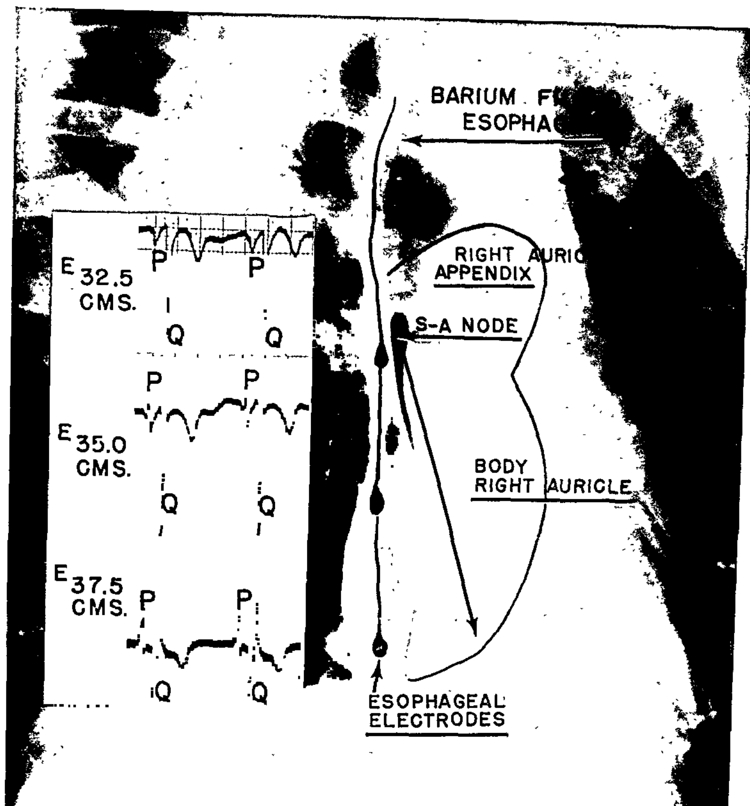
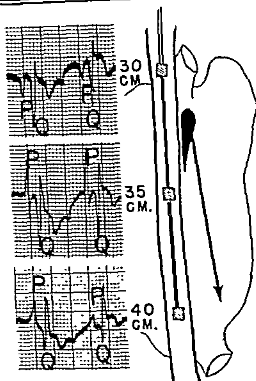


Figure 86 Electrocardiograms of normal chest with barium filled esophagus, diagrammed to show relative positions of esophageal electrodes and right auricle Esophageal leads graphically superimposed The right auricle and sino-auricular node, anterior and lateral to the esophagus, are in close proximity to the esophageal electrodes Configuration of the P waves is typical of the normal esophageal electrocardiogram *The tracing from the highest auricular* level exhibits a deep negative P wave (E 32 5 cm), mid-auricular levels (E 35 0 cm) a diphasic P wave is recorded, and at lowest auricular level (E 37 5 cm) large positive P waves are inscribed These deflections indicate that the impulse travels in a cephalocaudal direction, starting in the upper end of the auricle and moving toward the lower end



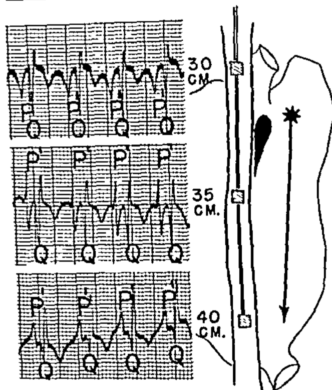


Figure 87 Esophageal leads from electrodes 30, 35 and 40 cm from the lips recorded simultaneously during tho-

cles (E 40) The impulse therefore travels in the usual cephalocaudal direction.

(B) Auricular paroxysmal tachycardia produced by stroking the right auricle at a site near the sino-auricular node The P' waves are negative at E 30, diphasic at E 35 and positive at E 40 These deflections are similar to the P waves of normal sinus rhythm and indicate that the impulse is traveling in a cephalocaudal direction.

at or above the level of the sino-auricular node with the result that the predominant deflection from such an electrode is negative. Conversely, the normal wave travels toward the caudal end of the auricles and the deflections from esophageal electrodes at successively greater distances below the node are progressively more positive.

Observation 8 Configuration of P' Wave in Esophageal Leads

Focus on the Cephalic End of the Auricle: Auricular paroxysmal tachycardia was produced in a patient undergoing operation in the thorax by stroking the right auricle at a site near the sino-auricular node. The P' waves of the tachycardia in esophageal lead electrocardiograms (Figure 87B) are similar to the P waves of normal sinus rhythm (Figure 87A); they are inverted in tracings from the level of the sino-auricular node, diphasic in tracings from the mid-auricular level, and upright in tracings from the caudal level of the auricles. Therefore, the impulse is traveling in a cephalocaudal direction during both the paroxysm of tachycardia and the normal sinus rhythm.

In a patient with mitral stenosis, auricular paroxysmal tachycardia was induced by exertion while electrodes were in the esophagus. The esophageal electrode at the level of the sino-auricular node recorded a diphasic P'

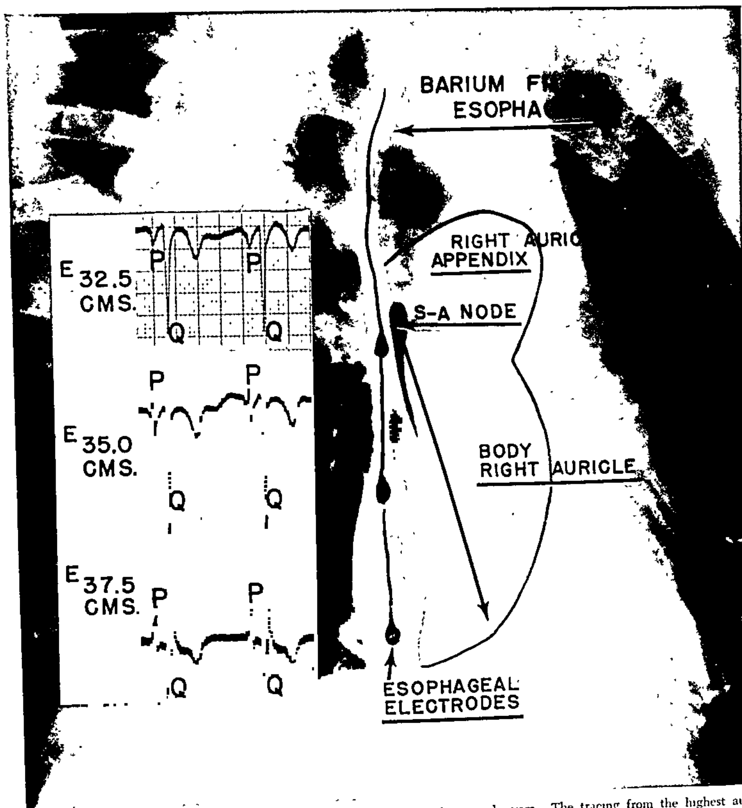
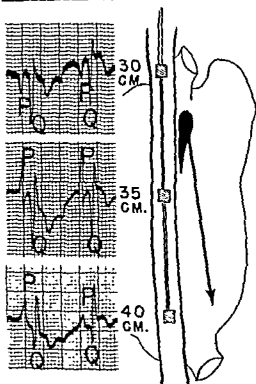


Figure 86 Electrocardiograms of normal chest with barium filled esophagus, diagrammed to show relative positions of esophageal electrodes and right auricle Esophageal leads graphically superimposed The right auricle and sino-auricular node, anterior and lateral to the esophagus, are in close proximity to the esophageal electrodes Configuration of the P waves is typical of the normal esophageal electrocardiogram The tracing from the highest auricular level exhibits a deep negative P wave (E 32 5 cm), mid-auricular levels (E 35 0 cm) a diphasic P wave is recorded, and at lowest auricular level (E 37 5 cm) large positive P waves are inscribed These deflections indicate that the impulse travels in a cephalocaudal direction, starting in the upper end of the auricle and moving toward the lower end



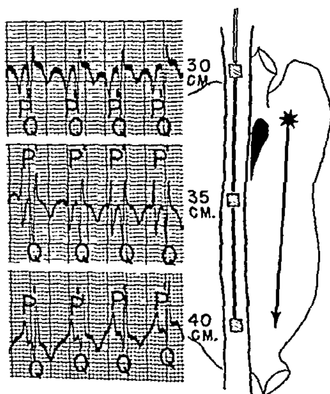


Figure 87 Esophageal leads from electrodes 30, 35 and 40 cm from the lips recorded simultaneously during thoracic surgery in a human subject

(A) Regular sinus rhythm The P wave is negative at the sino-auricular node (E 30), diphasic at the mid-auricular level (E 35) and upright at the caudal end of the auricles (E 40) The impulse therefore travels in the usual cephalocaudal direction

(B) Auricular paroxysmal tachycardia produced by stroking the right auricle at a site near the sino-auricular node The P' waves are negative at E 30, diphasic at E 35 and positive at E 40 These deflections are similar to the P waves of normal sinus rhythm and indicate that the impulse is traveling in a cephalocaudal direction

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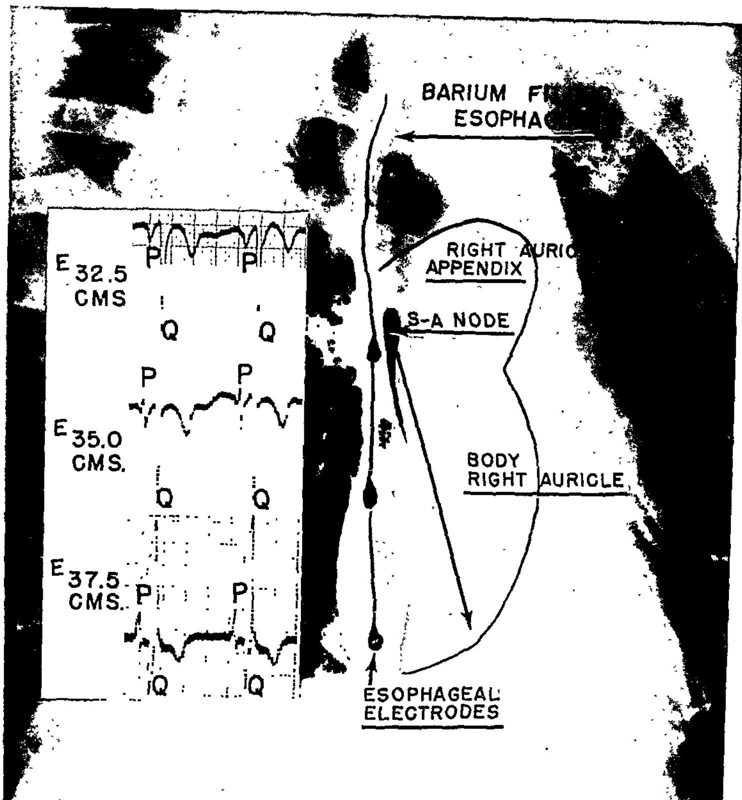


Figure 86 *Electrocardiograms of normal chest with barium filled esophagus, diagrammed to show relative positions of esophageal electrodes and right auricle Esophageal leads graphically superimposed The right auricle and sino-auricular node, anterior and lateral to the esophagus, are in close proximity to the esophageal electrodes Configuration of the P waves is typical of the normal esophageal electrocardiogram The tracing from the highest auricular level exhibits a deep negative P wave (E 32 5 cm), mid-auricular levels (E 35 0 cm) a diphasic P wave is recorded, and at lowest auricular level (E 37 5 cm) large positive P waves are inscribed These deflections indicate that the impulse travels in a cephalocaudal direction, starting in the upper end of the auricle and moving toward the lower end*

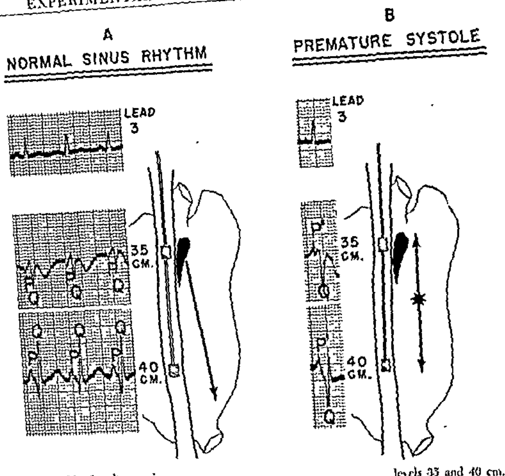


Figure 89 ... levels 35 and 40 cm. from the l... 40 it is positive The
(A) ...
impulse t... cephalocaudal direction.
(B) Auricular premature systole produced by stroking the *middle* of the right auricle The P′ wave is positive in both esophageal leads, indicating that the impulse arises at the mid-auricular level and travels toward both electrodes simultaneously.

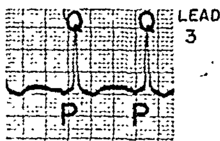
wave while the electrode at the lower auricular level recorded an upright P′ wave (Figure 88B). The configuration of these deflections indicates that the tachycardia impulse originated cephalad to the normal sinus beat and traveled in the same general direction.

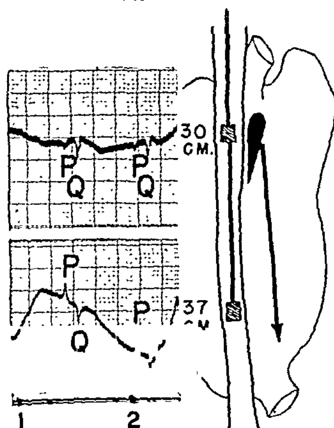
Focus on the Center of the Auricles: An auricular premature systole was produced by stimulation of a site on the mid-portion of the right auricle of a patient during thoracic surgery. Esophageal electrodes placed at high and low auricular levels both recorded upright P′ waves (Figure 89B), showing that the impulse arose *midway* between the two electrodes and traveled toward both auricular extremities simultaneously Similar deflections were obtained when auricular paroxysmal tachycardia was produced by stimulation at the center of the auricle. During normal sinus rhythm in the same patient, (Figure 89A) the P wave is inverted in the esophageal lead from the higher auricular level and upright in the lead from the lower auricular level, indicating that the normal impulse travels in a cephalocaudal direction.

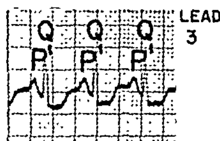
Focus on the Caudal End of the Auricle: An auricular premature systole (X in Figure 90A) was produced in man by stimulating the caudal region of the right auricle, three simultaneous esophageal leads were recorded. At the lowest auricular levels the P′ wave is inverted, at mid-auricular levels it is biphasic, and at high auricular levels it is upright. These deflections are

A
NORMAL SINUS RHYTHM

B
AURICULAR TACHYCARDIA







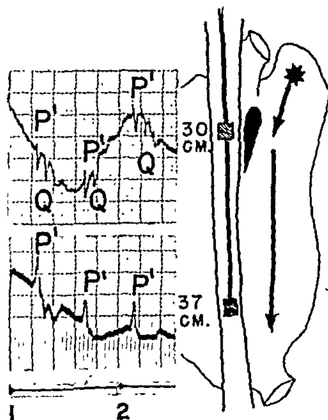
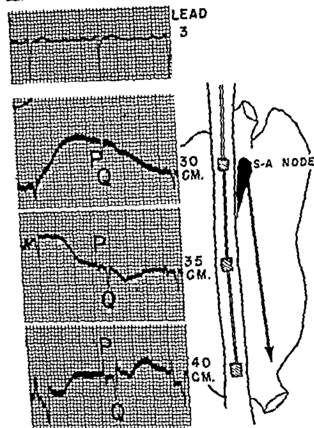


Figure 88 Patient with mitral stenosis in whom frequent attacks of auricular tachycardia were produced by exertion (*see Figure 43*)

(A) Simultaneous esophageal leads E 30 and E 37, and limb lead 3 recorded during normal sinus rhythm. The impulse travels from E 30 where a pure negative deflection is recorded to E 37 where the deflection is purely positive The P wave in lead 3 in inverted

(B) Same esophageal leads in same patient. Auricular tachycardia was induced by exertion (while electrodes were in esophagus) In E 30 the auricular deflection is biphasic indicating that the site of origin of the tachycardia impulse was cephalad to that of normal sinus rhythm

At E 37 large upright P' waves are seen



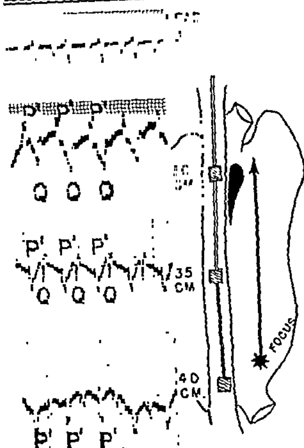


Figure 91 Simultaneous lead 3 and esophageal leads from electrodes 30, 35 and 40 cm from the lips in a patient undergoing thoracic surgery

(A) Regular sinus rhythm At E 30 the P wave is completely negative, at E 40 it is positive The impulse therefore travels in a cephalocaudal direction

(B) Auricular paroxysmal tachycardia produced by stroking the caudal end of the auricle The rate is 200 beats per minute Now the auricular deflection at E 40 is completely negative and at E 30 it is completely positive. Thus the impulse of tachycardia from a caudal focus travels in a caudocephalic direction, opposite that of the normal sinus beat

opposite in direction to the P waves of the normal beat (first two beats). Clearly, the experimentally produced impulse started at the caudal end of the auricle and traveled in a cephalic direction, or opposite the direction of the normal sinus beat Figure 90B shows a short run of auricular tachycardia produced in the same patient a few moments later and from the same focus as the premature systole Again the P' wave is inverted at the lowest, biphasic at the middle, and upright at the highest auricular level During this paroxysm of tachycardia the impulse obviously started at the stimulated point and traveled in a caudocephalic direction exactly as did the premature systole. The P' waves in esophageal and limb lead electrocardiograms are identical in the two disturbances.

Figure 91 shows another example of auricular paroxysmal tachycardia produced by stimulation of the caudal region of the right auricle. Again the configurations of the auricular deflections in the esophageal leads show that the impulse traveled in a caudocephalic direction

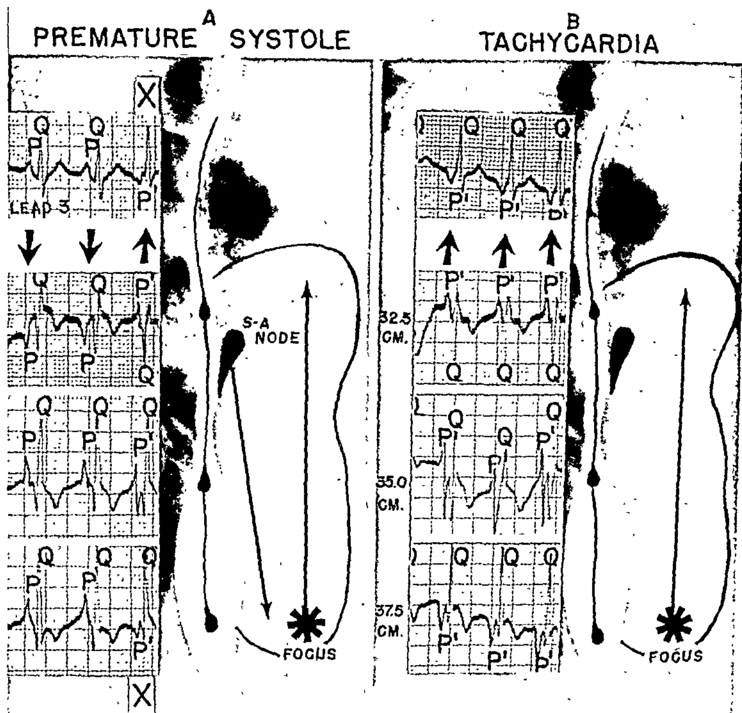


Figure 90. (A) Auricular premature systole (X) produced in right auricle of man by stimulation during surgery at site on caudal end of auricle marked "focus" Lead 3 and esophageal leads 32 5, 35.0 and 37 5 cm from lips recorded simultaneously. The P wave of the normal sinus beat (first two beats) is negative in the esophageal lead from the high auricular level, and upright in the leads from mid-auricular and low auricular levels, signifying that the excitation wave travels in a cephalocaudal direction

The P' wave inscribed during the premature beat *(third beat) is inverted in the esophageal lead from the* lowest auricular level, biphasic in the lead from the mid-auricular level and upright in the lead from the highest auricular level This signifies that the impulse starts at the lower end of the auricle and moves upward Thus the excitation wave of a premature beat from an ectopic focus low in the auricles travels in a direction opposite that of the normal sinus beat

(B) Same leads in same patient Shortly after the auricular premature systole was produced, the auricle was again stimulated at the same site and auricular tachycardia developed. The P' waves of the tachycardia are similar to those of the premature systoles and are opposite in direction to those of the normal beat

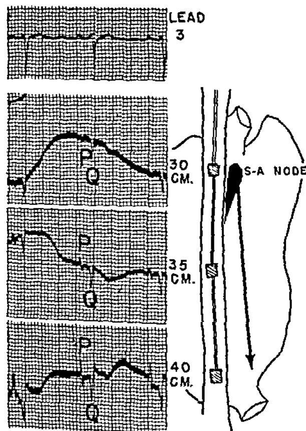
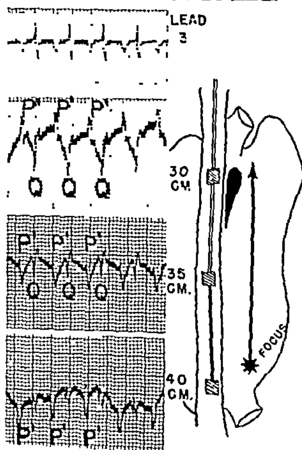


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(A) Regular sinus rhythm At E 30 the P wave is completely negative, at E 40 it is positive The impulse therefore travels in a cephalocaudal direction.



(B) Auricular paroxysmal tachycardia produced by stroking the caudal end of the auricle. The rate is 200 beats per minute Now the auricular deflection at E 40 is completely negative and at E 30 it is completely positive. Thus the impulse of tachycardia from a caudal focus travels in a caudocephalic direction, opposite that of the normal sinus beat

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Figure 91 shows another example of auricular paroxysmal tachycardia produced by stimulation of the caudal region of the right auricle. Again the configurations of the auricular deflections in the esophageal leads show that the impulse traveled in a caudocephalic direction

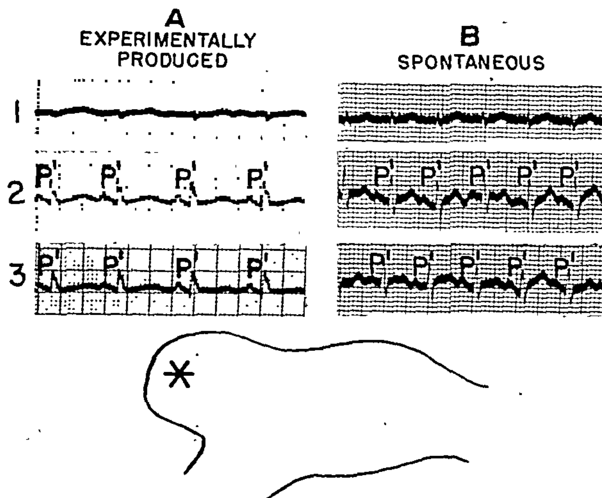


Figure 92. (A) Auricular paroxysmal tachycardia in man produced experimentally by stimulating the right appendix The P′ wave is upright in leads 2 and 3 and isoelectric in lead 1.

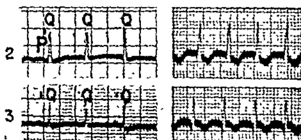
(B) Record from a patient with spontaneously occurring auricular tachycardia The P′ waves are identical to those from the experimental disturbance (A)

(Figure 91B) in contradistinction to the normal beat which ran in a cephalocaudal direction (Figure 91A).

The above observation, like observations 1 and 6, clearly shows the fundamental similarity between experimental auricular tachycardia and premature systoles in man. In esophageal lead electrocardiograms of both arrhythmias, a pure negative deflection is recorded over the ectopic focus. As the distance between the electrode and the focus increases, the positive deflections in the esophageal leads become larger. Since the esophageal lead electrocardiograms from a given auricular level are identical in auricular premature systole and auricular paroxysmal tachycardia from the same focus, the mode of origin and course of the excitation wave is identical in the two disturbances.

RELATIONSHIP BETWEEN CLINICAL AND EXPERIMENTALLY PRODUCED AURICULAR PREMATURE SYSTOLES AND AURICULAR PAROXYSMAL TACHYCARDIA

The similarity between the electrocardiographic deflections of experimentally produced and spontaneous auricular paroxysmal tachycardia in man is illustrated in Figures 92, 93 and 94. Figure 92A is a tracing of auricular tachycardia produced experimentally in man by stimulating the right appendix, Figure 92B shows spontaneous auricular tachycardia The P′ waves from leads 2 and 3 in the two records are upright and similar in appearance. Figure 93A shows tachycardia experimentally produced from the body of the right auricle; Figure 93B reflects the spontaneously occurring disturbance. In both these instances the P′



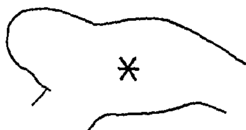


Figure 93 (A) Auricular paroxysmal tachycardia in man experimentally produced by stimulating the center of the [illegible]

[illegible] in man The P′ waves are isoelectric as in the experimentally produced disturbance

wave is difficult to identify. Figure 94A illustrates a paroxysm of tachycardia produced by stimulating the caudal end of the right auricle, Figure 94B is the electrocardiogram of a spontaneous disturbance. Here the P′ waves in the two records appear almost identical.

As noted in Chapter IX, reporting a study of 107 patients with tachycardia, the P′ waves of this arrhythmia in the standard limb leads fall into three relatively distinct classifications. upright, isoelectric, and inverted Our findings demonstrate that in man, tachycardias originating in the cephalic end of the auricles exhibit upright P′ waves, those arising near the center of the body of the auricle show isoelectric or low upright or inverted P′ waves, those starting near the caudal region inscribe deeply inverted P′ waves, especially if the P′-R interval is short. These conclusions are based upon identical results consistently found in 14 experiments in man and in 12 experiments in animals Furthermore, the same conclusion regarding the relationship between the site of the ectopic focus and the direction of the auricular deflection is reached by calculation of the total auricular electrical axes (Observation 2).

Esophageal electrocardiograms have been taken during spontaneous auricular premature systole in six patients and during spontaneous paroxysmal tachycardia in three patients. In all instances these records were identical with esophageal leads taken during experimentally produced auricular premature systoles and paroxysmal tachycardia in man (Figure 90). From such studies, the unequivocal conclusion may be drawn that the excitation waves of both spontaneous and experimentally produced

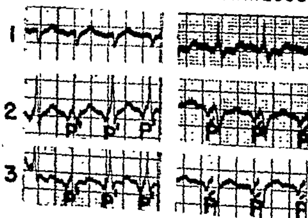




Figure 94 (A) Auricular paroxysmal tachycardia in man experimentally produced by stimulating the caudal part of the right auricle The P′ waves are inverted in leads 2 and 3.

(B) Auricular paroxysmal tachycardia occurring spontaneously in man. The P′ waves are almost identical to those from the experimentally produced disturbance.

auricular premature systoles and paroxysmal tachycardia originate at an ectopic focus and pursue an identical course: Waves arising at the caudal end of the auricles travel in a caudocephalic direction; those arising at the cephalic end travel in a cephalocaudal direction; those arising at the center of the auricles travel toward both extremities simultaneously. No evidence of a circus movement has been found in either disturbance.

The concept of the unitary nature of the auricular arrhythmias is discussed in detail in Chapter XVIII. The first stage in the development of this concept is the establishment of the relationship of auricular premature systole to paroxysmal tachycardia. The resemblance between these disturbances is shown by the identity of their P' waves in limb and esophageal leads when they arise from the same ectopic focus. Their close relationship is further emphasized by the fact that either arrhythmia can be produced with ease in human subjects by mechanical stimulation of the auricles. We have repeatedly produced single premature beats in both man and animals while stimulating the auricles mechanically. Occasionally two successive premature beats occur, in other instances three, four, five and six successive "premature" beats appear, constituting a short paroxysm of tachycardia. It is thus clear that the essential difference between auricular premature systoles and auricular paroxysmal tachycardia is the rate of discharge from the ectopic focus. In tachycardia the ectopic focus discharges more rapidly than the sinus node and usurps the pacemaking function. In premature systole only occasional beats arise from the ectopic focus and the sinus rhythm remains dominant.

Sir Thomas Lewis [397] came to these same conclusions regarding the relationship between auricular premature systole and paroxysmal tachycardia with the relatively primitive tools at his disposal coupled with excellent clinical observations and deduction.

SUMMARY AND CONCLUSION

Auricular premature systole and auricular paroxysmal tachycardia have been produced experimentally in 18 patients by mechanically stimulating various parts of the auricles during cardiac and pulmonary surgical procedures. The resulting arrhythmias were studied by means of high-speed cinematography and by limb and esophageal lead electrocardiography.

The cinematographic appearance of experimentally produced auricular premature systole and auricular paroxysmal tachycardia in man is identical with that observed in the dog. In both arrhythmias, whether in man or in the animal, the contraction wave arises at the site of stimulation and spreads outward in all available directions.

If auricular premature systole and auricular paroxysmal tachycardia are produced from the same ectopic focus in a patient, the configuration of the P' waves in limb lead electrocardiograms is identical in the two disturbances. If the focus is at either appendix, the P' wave is upright in leads 1, 2, 3, and AVF, and similar to the normal P wave; the impulse travels from the appendix in a cephalocaudal direction as calculated from the total auricular electrical axis. If the impulse originates in the center of the auricle, it travels toward both auricular extremities; the P' waves are then isolectric or small upright or inverted in leads 1, 2, 3 and AVF. When the impulse starts at the caudal region, it travels in a caudocephalic direction, the P' waves are inverted in leads 2, 3 and AVF The P'-R interval in either auricular premature systole or auricular paroxysmal tachycardia from the caudal region is often shorter than normal and may be as short as 0.08 second. If the ectopic focus is at the cephalic extremity, the P'-R interval in the two arrhythmias may or may not be prolonged.

Esophageal lead electrocardiograms from high, middle and low auricular levels were recorded simultaneously in 10 patients during experimentally produced auricular premature systole or auricular paroxysmal tachycardia.

When the caudal end of the auricles is stimulated, the deflection from the low auricular level is negative, the deflection from the mid-auricular level is biphasic, and the deflection from the high auricular level is positive; the excitation wave therefore travels in a caudocephalic direction. If the cephalic end of the auricles is stimulated, the deflections in the esophageal leads are opposite in direction to those recorded when the ectopic focus is at the caudal end, indicating that the excitation wave pursues a cephalocaudal course.

The P' waves of clinical and experimental auricular premature systole and auricular paroxysmal tachycardia are identical in both esophageal and limb lead electrocardiograms. In either arrhythmia, whether it occurs spontaneously or is produced experimentally in man or animal, the excitation wave starts at an ectopic focus and travels outward in all available directions. There is no evidence of a circus movement.

CHAPTER V

The Motion of the Auricles in Auricular Flutter

AURICULAR flutter is a condition characterized by a rapid, regular auricular rate within a range of 150 to 360 beats per minute, usually between 260 and 320 beats per minute. Auriculo-ventricular block resulting in a substantially lower ventricular rate is almost invariably present. Most commonly the block is in a ratio of 2:1 with a ventricular rate of 130 to 160 beats per minute; higher degrees of block may occur with ventricular rates of 65 to 80 beats per minute (4:1); occasionally, complete auriculo-ventricular dissociation is present with an independent ventricular rate of 30 to 40 beats per minute, rarely, auriculo-ventricular block is absent, in which case the ventricular rate is the same as that of the auricle (1:1).*

The nature and mechanism of auricular flutter are discussed in detail in Chapters VI and VII.

CLINICAL CONSIDERATIONS

Although in a broader sense it may be stated that auricular flutter occurs under the same conditions which are known to favor the development of auricular fibrillation, certain significant differences are to be noted.

General Incidence: Auricular flutter is much less common than auricular fibrillation. The ratio of the former to the latter, as reported for large series of cases in various clinics, ranges between 1 to 10 and 1 to 20.[65, 345, 481, 562]

Age and Sex Incidence: Although auricular flutter can occur at any age, it is a disease essentially of middle and advanced years. This arrhythmia is four or five times more frequent in males than in females. Since its occurrence is predominantly in the elderly, its association with degenerative cardiovascular disease is relatively frequent.

Pathology: As in auricular fibrillation, no specific pathologic anatomy is recognized for auricular flutter.

Symptomatology: The almost invariable presence of auriculo-ventricular block with a consequent slowing of the ventricular rate tends to mitigate the effects of auricular flutter. In the absence of serious myocardial disease, few symptoms may develop even when the disorder persists for many months. Sir Thomas Lewis cites the case of a patient "in whom the ventricle has continued to beat at 140 per minute night and day for ten years, his condition being much the same at the beginning and end of this period. Such cases illustrate the resistance of a relatively healthy heart to prolonged and considerable strain in a most convincing manner."[390] When the arrhythmia occurs in short paroxysms the symptoms are largely subjective, namely, palpitation, apprehension and perhaps slight breathlessness.

On the other hand, on a background of serious myocardial disease, the arrhythmia may rapidly precipitate severe congestive failure. In the rare instances in which auriculo-ventricular block is absent, a rapid ventricular rate of 300 or more beats per minute may bring about complete collapse and unconsciousness even in an otherwise relatively normal heart. Similarly, a state of collapse may occur in a patient exhibiting flutter with 2:1 or higher-grade

* It is questionable that 1 1 flutter is basically distinguishable from rapid auricular paroxysmal tachycardia (Chapter IX).

auriculo-ventricular block when, as a result of emotional stress or other factors, the block disappears and the ventricular rate suddenly doubles or quadruples.

Diagnosis: Although a final and definite diagnosis can be made only by means of the electrocardiogram, bedside recognition of auricular flutter is not infrequently possible. A persistent, regular tachycardia at a constant rate within the range of 130 to 160 beats per minute in a middle-aged or elderly person is highly suggestive of flutter; the possibility is further strengthened by a history of shorter paroxysms associated with palpitation or fainting In such an instance carotid sinus pressure serves to differentiate auricular flutter from sinus tachycardia and from auricular and ventricular paroxysmal tachycardias. If this procedure results in significant slowing of the pulse rate or the dropping out of *several beats, followed* by a "jerky" retreat to the previous rate after cessation of pressure, the diagnosis of flutter can be regarded as almost certain. In sinus tachycardia, carotid sinus pressure may slow the rate slightly but not significantly In auricular paroxysmal tachycardia, it either terminates the bout with sudden restoration of normal sinus rhythm, or is entirely ineffective. In ventricular paroxysmal tachycardia, carotid sinus pressure is without effect. It should be borne in mind that ventricular paroxysmal tachycardia normally tends to be slightly irregular, after a number of perfectly regular beats slight interruptions in rhythm may occur spontaneously Care should be taken *not to* attribute such interruptions to carotid sinus pressure (see Table I)

Other clinical procedures which may aid in the diagnosis of auricular flutter are:

(1) *Change in intensity of the first heart sound* This is due to variations in the P-R intervals resulting from slight differences in the ventricular response commonly present in flutter (Figure 95). When the P-R interval is prolonged the first sound is usually diminished The prolongation need not be great. "Even an interval of 0 21 second or 0.22 second will produce a faint first sound . . . Conversely, as one might suspect, when the P-R interval is short, the first sound is increased." [264]

(2) *Effects of exercise.* (A) In auricular flutter, especially with the higher-grades of blocks (4:1, etc.), exercise may suddenly bring about an exact doubling of the original regular ventricular rate, as from 65 or 70 to 130 or 140 beats per minute. (B) In other instances, exercise may result in a sudden change from a slow, absolutely regular ventricular rate to a rapid, irregular rate. (C) When the original ventricular rate is *relatively slow* and irregular because of an irregular block, exercise will sometimes cause the rate *to greatly increase* and become completely regular. Changes of type C differentiate auricular flutter with irregular block from auricular fibrillation in which exercise also brings about an increase in rate but causes the rhythm to become more irregular. Under all three circumstances, cessation of exercise will be rapidly followed by reversion to the original rate *and rhythm.*

(3) *Pulse rate.* A pulse rate substantially above 180 (190 to 240) rarely occurs in *auricular flutter*, for, as pointed out by Harvey and Levine [264] "if the rate is 190 to 200 and perfectly regular, it is extremely unlikely that flutter is present. The auricular rate would of necessity be either 190 to 200 with a 1:1 rhythm or 380 to 400 with a 2:1 block The former possibility can be fairly well ruled out because 1:1 flutter is extremely rare and the auricular rate of 190 is too slow (if *untreated*). The latter is very unlikely because the auricular rate in flutter rarely exceeds 360."

(4) *Effect of deep breathing.* The effect of deep inspiration is similar to though less striking than that of carotid sinus pressure. In flutter this procedure often results in a slowing of the rate and causes the rhythm to become more irregular than in sinus tachycardia or auricular and ventricular paroxysmal tachycardias.

(5) *Visible flutter waves in the neck veins.* Occasionally, in some instances of high-grade block when the ventricular responses are infrequent, small rapid regular waves transmitted

TABLE I

DIFFERENTIAL DIAGNOSIS OF THE REGULAR TACHYCARDIAS

	Auricular Flutter	*Paroxysmal Auricular Tachycardia*	*Paroxysmal Ventricular Tachycardia*	*Sinus Tachycardia*
Onset and Termination	Sudden	Sudden	Sudden	Very Gradual
Duration of Attacks	Days, weeks, months	Minutes, hours, days	Minutes, hours, days	Depends on underlying cause
Average Rate	130 to 160	160 to 240	160 to 180	Rarely above 140
Rhythm	Usually completely regular	Completely regular	Usually slightly irregular	Usually completely regular
Effect of Carotid Sinus Pressure	Grossly but only temporarily affected	Completely abolished or no effect at all	Usually no effect	Slight but insignificant temporary slowing
Effect of Exercise	May be pronounced (such as doubling of rate) or no effect at all	No effect	No effect	Usually significant increase in rate
Variations in Intensity of First Heart Sound	Present	Absent	Present	Absent

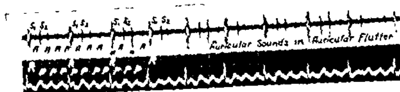


Figure 95 Simultaneous phonocardiogram and electrocardiogram from a patient with auricular flutter. Note the unequal intensity of the auricular sounds in the phonocardiogram during flutter The P' wave deflections in the simultaneous electrocardiogram are all identical (From Levine & Harvey· *Clinical Auscultation of the Heart*, page 87, Courtesy W B Saunders Co, 1949)

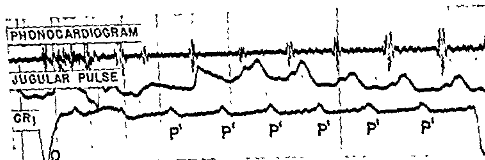


Figure 96 Simultaneous phonocardiogram, jugular pulse tracing and electrocardiogram (CR_1) from a patient with auricular flutter *Note the absolute constancy of the P' waves with the marked variation of the sounds and of the jugular pulsations.* Both this illustration and Figure 95 demonstrate that in human auricular flutter there is mechanical variation from beat to beat although the electrical activity remains constant. (Courtesy Howard B Burchell, M.D)

from the auricles may be seen in the veins of the neck as minute undulations.

(6) *Duration of attacks.* Paroxysms of auricular flutter usually persist for long periods of time, for months or even years This is in sharp contrast to sinus and auricular paroxysmal tachycardias in which the paroxysms usually terminate within a period of hours or days.

Prognosis: The outlook for patients with auricular flutter depends primarily upon the state of the myocardium and secondarily upon the response to treatment When the myocardium is relatively healthy and response to therapy good, the prognosis is excellent, even if the arrhythmia tends to recur, with proper management the patient can enjoy satisfactory health for indefinite periods of time. However, when the myocardium is seriously damaged and the arrhythmia resistant to therapy, the outlook is unfavorable; the added burden of the persistent tachycardia will tend to precipitate severe congestive failure

EXPERIMENTAL STUDIES OF AURICULAR FLUTTER

The term "flutter" was first used to characterize auricular contractions in 1887 when MacWilliam[422] applied it to an experimentally produced disturbance in the auricles of animals. Presumably the term was adopted because of a resemblance of the motion of the auricles to the fluttering wings of a bird. MacWilliam suggested that the rapid, rhythmic contractions were caused by a series of impulses from a single ectopic focus. No electrocardiograph was available at that time, and the exact nature of the disturbance observed by MacWilliam remained unknown. Ritchie,[305] in 1905; Gibson,[237, 238] in 1905, 1906; and Hertz and Goodhart,[275] in 1908, directed attention to a similar arrhythmia in man. Jolly and Ritchie,[296] in 1911, first applied the term of auricular [illegible]
to the [illegible]

[illegible] At the turn of the cen-

Figure 97. Reproduction of the results of Lewis' most complete experiment, from these determinations he calculated the course of the flutter wave and concluded that the circus movement exists According to the circus movement theory, the impulse travels up the right auricle and down the left and thus moves in an opposite direction in each. No determinations are recorded on the long downward path over the left auricle (After Lewis *The Graphic Registration of the Heart Beat*, courtesy Shaw and Sons, Ltd., London.)

tury, the mechanism of auricular flutter was the subject of investigation by many workers.[164, 165, 522] The arrhythmia was generally considered to result from a discharge of stimuli from a single or multiple ectopic foci. During this period a number of investigators advanced the concept of "circulating rhythms." Mayer,[445] in 1908, produced circulating rhythms in rings cut from the bell of a medusa. Mines,[455, 456] in 1913, 1914, obtained unidirectional contraction waves in ring preparations cut from the auricles of large rays. Garrey,[217] in 1914, produced circulating contractions in rings cut from the base of the ventricles of large turtles. Mines[456] and Garrey[217] suggested that such rhythms could be responsible for the condition known as fibrillation. It was the work of these investigators that formed the basis for the monumental contributions of Sir Thomas Lewis and his associates[363, 364, 365, 366, 367, 368, 369, 370] from 1918 to 1921.

The Circus Movement Theory: The prevailing theory of the nature of flutter was advanced by Lewis and his co-workers and has dominated medical thinking on this subject for more than 30 years. According to Lewis' theory, a wave of contraction travels through both auricles in a unidirectional circus path (probably counter-clockwise) around the mouths of the two venae cavae (Figure 97). The main or "mother" wave moves in a narrow muscle path and, in its course, gives rise to "daughter" waves which spread over the remainder of the auricular bodies and appendices. This concept has been accepted by most clinical and experimental workers and is included as established fact in nearly all standard textbooks.

The evidence upon which Lewis based his theory is illustrated in the following summary of his most complete experiment.[366, 385]

In a dog exhibiting electrically induced flutter, the exposed heart was explored electrographically by means of paired electrodes placed directly on various parts of the auricles. From the time of onset of intrinsic deflection as determined by means of a simultaneously recorded lead 2, the course of the cardiac impulse through the auricles was calculated (Figure 97).

As shown in Figure 97, from the ectopic point of origin ("S") the course of the impulse was traced by determining the time of its arrival at successive points on the auricle between the caudal end and the appendix. Almost all determinations were made on the right auricle; only two observations were made on the left auricular appendix and none on the body of the left auricle. From the time values of 0.075 and 0.137 second obtained at the superior and inferior venae cavae, respectively, Lewis concluded that the impulse circled the superior vena cava, descended through the body of the left auricle, and embraced the inferior vena cava to re-enter the original circus path. In the absence of complete determinations the wave was assumed to travel through both auricles at a constant rate; on that basis, Lewis computed the time required for the impulse to return across the body of the left auricle to the 0.000 time-point. The result of his calculation corresponded almost exactly to the time of arrival of the second impulse as demonstrated electrocardiographically.

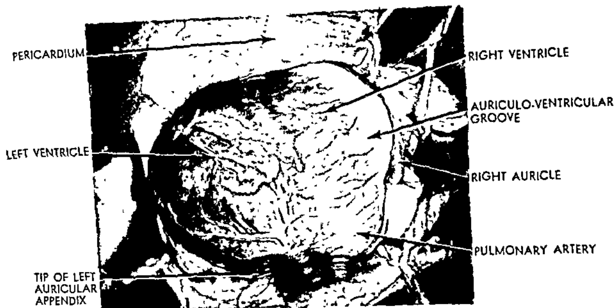


Figure 98 Illustration to demonstrate the type of exposure of the heart as utilized by Sir Thomas Lewis The heart is supported by a pericardial hammock sewed to the margins of the incision Note how poorly the auricles are exposed. See Figures 1, 2, 319 and 320 to compare the excellent exposure of the auricles obtained by the type of dissection employed in the present studies

This circumstance seems to have provided considerable support for the circus movement theory That Lewis himself was aware of the significance of the gap in his determinations is indicated in the following statement· "No doubt remains as to the course taken by the excitation wave during the flutter in the experiments which have been discussed, insofar as the exposed surface of the right auricles, the left surface of the superior cava, the band and left appendix are concerned. The weakness obviously lies in the length of the gap between the intra-auricular band and the inferior cava on the return journey"* [364]

Lewis believed that clinical evidence confirming the circus movement theory could be derived from the following observation. In a patient with auricular flutter, he and his associates [372] recorded three simultaneous leads from frontal, sagittal and horizontal planes of the body. By detailed examination of the momentary atrial electrical axes, they determined that the axis revolved through 360 degrees during the course of each auricular cycle. This revolution was attributed to the movement of the main excitation wave and was regarded as proof that the wave pursued an elliptical path around the cavae. Decherd, Ruskin and Herrman, in 1945,[135] and Grishman and co-workers, in 1950,[240] confirmed Lewis' findings and supported his conclusion.

Recent Experimental Studies: During the 30 years following Lewis' classic observations on the mechanism of auricular flutter, various authors have investigated this subject. Up to the present, no facts have been submitted which conclusively prove the circus movement theory, and no convincing evidence has been advanced in favor of an alternative concept. All previous and subsequent studies of auricular flutter were

* In his experiments on dogs, Lewis generally exposed the heart by making parallel incisions to the right and left of the sternum near the nipple lines The heart was held in a "pericardial swing" made by splitting the pericardial sac and sewing the cut edge of each piece to the chest wall on the corresponding side This [illegible]
exposure of the
but did not exp
demonstrates the
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less extensive than those of Lewis and less direct in approach.

Brams and Katz;[64] Scherf and Boyd;[541] and Wilson;[636] object to the clinical evidence advanced by Lewis on the grounds that the course of the impulse in a narrow band of muscle cannot be deduced from the form of the auricular deflections. It is generally acknowledged that the auricular deflections represent electrical activation of the entire auricular musculature. According to the circus movement theory, the main mass of the auricular musculature is activated by the daughter waves which spread in all directions from the mother path. Therefore, these authors maintained, activation of the narrow mother path alone would scarcely produce a deflection on the electrocardiogram; hence, the auricular deflections cannot represent the movement of the impulse in a circus pathway. Other objections in this phase of Lewis' work are presented in Chapters VII and VIII.

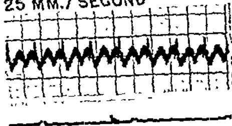
Brams and Katz[64] produced flutter in dogs by the post-electrical stimulation method and, with large forceps, effected a severe crush in Lewis' circus pathway, flutter continued uninterrupted. These authors admitted that, although the original path described by Lewis was completely severed, the possibility of two new daughter circus movements, one on each side of the crush, could not be ruled out. This opinion was shared by Wiggers.[648]

Weiner and Rosenbleuth,[623] and Selfridge,[554] after a detailed and highly technical mathematical analysis of the fundamental properties of auricular muscle in flutter, pointed out certain inconsistencies in Lewis' calculations pertaining to circus movement; nevertheless, they concluded that the circus movement of active waves was probably the mechanism of auricular flutter. From their data they could neither prove nor disprove other possibilities, such as (1) one or more active auricular pacemakers; and (2) a continuous external stimulus causing the most excitable region or regions to become active auricular pacemakers.

In later publications, Rosenbleuth and Garcia Ramos[516, 517, 519] completely accept the circus movement theory. They blocked the intercaval auricular bridge by crushing or by application of cocaine, and believed that this tended to perpetuate the circus movement along its original path by preventing the short-cuts which might terminate the arrhythmia; such procedures also were considered to facilitate the production of flutter. In another experiment they extended the obstacle by further application of cocaine in order to widen the circus path; this resulted in a decrease in the flutter rate, interpreted as indicating that a longer time was needed for the impulses to travel around the widened perimeter of the hypothetical circus path.

The contributions made by Scherf and his associates are perhaps the most illuminating since those of Lewis. These workers were the first to produce auricular arrhythmias by direct application of aconitine to the auricular wall; a modified form of their technique has proved superior to all other methods employed in our laboratory. They further demonstrated that a reversal of the arrhythmias could be obtained by cooling or clamping off the aconitine focus.[542] The mere facts that an arrhythmia can be initiated by local application of aconitine and terminated by cooling or clamping off the aconitine focus do not establish the nature of the disturbance. Rather, the value of the aconitine method is that it provides a reliable means of producing arrhythmias for purposes of study. Finally, Scherf and his associates have been among the few who have consistently maintained that the existence of a circus movement, as Lewis himself admitted, has never been conclusively established.

Scherf[543] produced flutter by injecting aconitine into the auricle and attempted to interrupt the circus path by placing ties across the taenia terminalis, in 16 of 17 experiments the flutter continued unchanged. Unfortunately, Scherf's investigations, like those of other workers' after Lewis, were based upon indirect methods of observation. Without the use of direct auricular leads or some other means



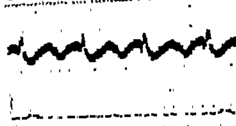


Figure 99 2.1 auricular flutter (produced by aconitine).
The tracings demonstrate the appearance of 2.1 auricular flutter in lead 2 recorded at normal (25 mm per second) and fast (50 mm. per second) speeds. Note that the typical saw-tooth appearance when recorded at a normal speed has a rounded appearance when the paper moves faster under the stylus (50 mm. per second). Electrocardiograms of this type were present when the motion pictures were taken

of direct observation, it is impossible to determine exactly what is happening in the auricle. Thus while some of the indirect evidence obtained by Scherf appeared inconsistent with the circus movement theory, the conclusions he could draw from his investigations of necessity were not decisive and often not consistent. In 1928 he wrote that "flutter and fibrillation are due to a very rapid stimulus formation and that a complete separation from other tachycardias is not justified"[538] More recent observations led him to a different conclusion, namely, that the different responses to vagal stimulation of auricular tachycardia and auricular flutter indicate "that we are dealing with two different types of stimulus formation." From these observations he suggests that the stimuli in flutter are continuous while in tachycardia they are intermittent[549] Such a theory has not received support from our observations. By 1950, Scherf and his associates had decided their earlier theory of fibrillation did not always hold true, for they stated: "The experiments are evidence which tends to support the theory that in some forms of auricular fibrillation more than one centre of rapid stimulus formation is active."[542] Among Scherf's objections to the circus movement theory is the statement that the auricular deflections of flutter resemble those of normal sinus rhythm This statement is invalid, since in some 80 per cent of cases the flutter deflection is opposite in direction to the normal P wave (Chapter VIII). That Scherf was unable to draw definitive conclusions from his indirect observations is shown by his statement in 1948 that ". . . these objections and the experiments discussed in this communication do not prove that a circus movement does not exist."[545]

MATERIALS AND METHODS

The weakness in the evidence presented by Lewis as well as by all workers who have expressed doubt concerning the circus movement theory lies in the fact that none has been able to trace the course of the flutter wave in its entirety. Without such direct observations, no final conclusions are possible. As described in the Appendix, the technique of dissection used in this laboratory permits complete exposure of both auricles.

Auricular flutter was produced in 40 dogs and photographed at camera speeds of 100, 750, 1000 and 2000 frames per second. In each instance the presence of flutter was confirmed by electrocardiograms taken simultaneously with the cinematographs (Figure 99). A given arrhythmia was included in the study only if it exhibited the recognized electrocardiographic criteria for the diagnosis of flutter, namely,



Figure 100. Photograph made with the heart lifted ventrally to demonstrate the relationship of the posterior surface of the heart and site of aconitine application. Aconitine was applied to the natural crevice formed by the inferior vena cava and a branch of the pulmonary vein.

regularly recurring waves of typically undulating configuration occurring at a rate of approximately 300 per minute, absent or short isoelectric periods, and 2:1 or greater auriculoventricular block. By means of slow motion pictures of both the left and the right auricles, the entire course of the contraction wave of auricular flutter could be directly visualized. By means of direct auricular leads from both auricles, the entire course of the excitation wave of auricular flutter could be accurately traced. The results of the cinematographic studies are discussed in this chapter. The corresponding electrocardiographic observations are presented in Chapter VI.

Methods of Production: Experimental auricular flutter was produced by three methods. (1) post-electrical stimulation (Lewis), [304] (2) intercaval block (Rosenbleuth and Garcia Ramos), [515, 516, 517, 519] and (3) aconitine (Scherf) [542, 543]

(1) The auricles were stimulated for various periods of time with single induction shocks from a pulse generator at rates of approximately 400 to 500 per minute. Flutter was readily produced during the period of electrical stimulation, only on rare occasions did the arrhythmia persist after stimulation had been discontinued for a length of time sufficient to permit adequate cinematographic study.

(2) Rosenbleuth and Garcia Ramos found that crushing or blocking of the intercaval bridge with cocaine facilitated the production of post-stimulatory auricular flutter, only rarely, and with great difficulty, could they obtain the arrhythmia without such block. In the present

A. Full diastole – star represents ectopic focus. Flutter was produced by applying aconitine at focus.

B. Early systole – contraction wave traveling toward appendix.

C. Appendix still in systole – rest of auricle in diastole. Note relaxation travels in same order as systole.

Figure 101. Enlarged photographs of auricular flutter produced by the application of aconitine to the caudal end of the auricle near the inferior vena cava. Successive pictures demonstrate that the contraction wave spreads across the auricle as a broad band from the site of origin near the inferior vena cava, through the body onto the right appendix. This is followed by a relaxation wave which follows an identical course. Flutter produced by post-electrical stimulation has the same appearance as that produced by aconitine.

study, intercaval block was produced in a few experiments by (a) crushing, (b) cocainization, or (c) searing with a hot glass rod. These procedures in our hands only moderately increased the ease with which flutter could be secured as a post-stimulatory effect.

(3) Aconitine proved the most effective agent in the production of flutter, the resultant arrhythmia usually was of sufficient duration to permit adequate photographic and electrocardiographic study. Early in the study, 0.05 cc. of a 0.05 per cent solution was injected into the wall of the auricle as recommended by Scherf. Later we found that topical application of aconitine was superior and easier to control; a small cotton swab saturated with 0.05 to 2.0 per cent solution was applied to an area 1 to 2 millimeters in diameter on the surface of the auricle. The chosen site was swabbed dry before the application of aconitine and the nearby region was packed with cotton to protect the ventricles from contact with the drug. The top-

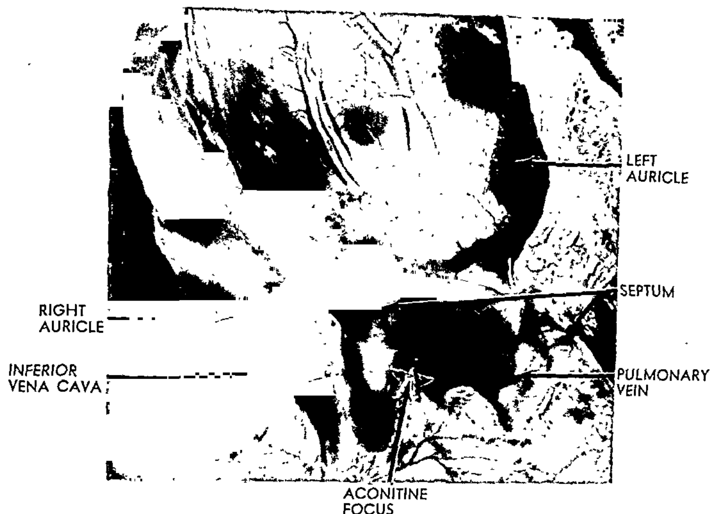


Figure 100 Photograph made with the heart lifted ventrally *to demonstrate the relationship of the posterior surface* of the heart and site of aconitine application Aconitine was applied to the natural crevice formed by the *inferior vena cava and a branch of the pulmonary vein.*

regularly recurring waves of typically undulating configuration occurring at a rate of approximately 300 per minute, absent or short isoelectric periods, and 2:1 or greater auriculoventricular block. By means of slow motion pictures of both the left and the right auricles, the entire course of the contraction wave of auricular flutter could be directly visualized. By means of direct auricular leads from both auricles, the entire course of the excitation wave of auricular flutter could be accurately traced. The results of the cinematographic studies are discussed in this chapter The corresponding electrocardiographic observations are presented in Chapter VI.

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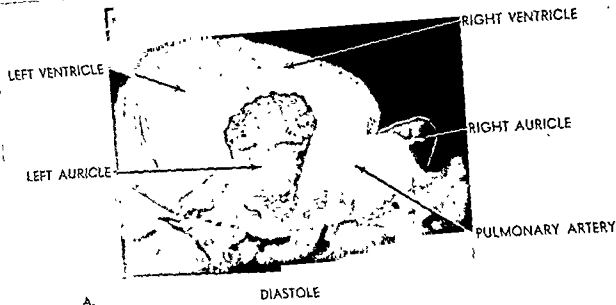




Figure 102 Enlargement of photographs with the camera viewing an area midway between the left and right auricles so that both could be visualized at the same time. *Flutter is* present

Aconitine was placed midway between the auricles in a natural crevice between the inferior vena cava and pulmonary vein. In A the auricles are in full *diastole*. In B the onset of auricular systole has just occurred Note both auricles begin to contract simultaneously. If auricular flutter were a circus mechanism the excitation wave should reach one auricle a significant period of time before it reached the other.

ical use of aconitine is preferable since it obviates the danger of accidental injection of the drug into the blood stream with resultant ventricular fibrillation. The most favorable site for the production of flutter by means of aconitine was found to be an area on the line of the caudal attachment of the inter-auricular septum; here a natural crevice is formed by the walls of the inferior vena cava and the pulmonary vein stemming from the lower lobe of the left lung. Application of the drug at this site produced flutter in 25 of 30 consecutive experiments (Figure 100).

THE CINEMATOGRAPHIC APPEARANCE *OF EXPERIMENTALLY PRODUCED* AURICULAR FLUTTER

Observation 1: Course of the Contraction Wave of Flutter from a Focus at the Caudal End of the Right Auricle

Flutter was produced by post-electrical stimulation or local application of aconitine at the caudal end of the auricle near the inferior vena cava (the site generally employed by Lewis and co-workers[364]) in 10 dogs, motion pictures of the right auricle were taken at 2000 frames per second. The cinematographic appearance of the flutter wave was the same regardless of the method used in its production. On the films, the following characteristics were apparent.

The contraction wave spread across the auricle as a *broad* band from the site of origin near the vena cava, through the body, into the right appendix. This was followed by a relaxation wave which pursued an identical course. Succeeding contraction waves followed each other rapidly, they often were initiated before diastole had reached all areas distant from the ectopic focus. (Figure 101)

From this experiment, it is easily understood that Lewis assumed the flutter wave pursued a unidirectional course, so far as the right auricle is concerned, when the ectopic focus is located at the caudal end of the auricle, the wave can be traced cephalad, from "right to left," to the appendix. However, the cinematographs clearly show (1) that the wave is not a narrow band, but *involves the entire width of the auricle;* (2) that each succeeding wave starts at the ectopic focus and does *not* come up from behind the inferior vena cava; (3) that as the wave passes over the superior vena cava *it continues in the same direction into the right appendix and does not turn down toward the* left auricle as it would if it were pursuing a circus path. The main path of the wave is the *only one seen; nothing is present to suggest a* "mother" path giving off "daughter" waves. Lewis[395] observed that as the auricular rate became faster, the speed of propagation of the contraction wave became slower. Cinematographic examination of the contraction wave appears to confirm this observation; the wave definitely travels more slowly in flutter than in normal sinus rhythm.

This observation casts considerable doubt on the circus movement theory and disproves some of its essential premises. It does not, however, completely exclude that concept.

Observation 2. Speed of the Contraction Wave of Auricular Flutter

Using the same technique as in the above experiment, an attempt was made to check Lewis' computations of the time required for the wave to traverse the complete circus pathway starting at the caudal end of the right auricle. Auricular flutter, occurring as an aftereffect of electrical stimulation of the right auricle at a point near the inferior vena cava, was photographed at 2000 frames per second. On the films, the wave could be seen clearly and the time consumed in its journey up the right auricle accurately determined. Generally, the distance from the inferior vena cava to the superior vena cava was traversed in nine seconds, cinematographic time (0.072 seconds actual time). Since the distance of the return journey in the left auricle from the superior vena cava to the site of origin at the caudal end is about the same, and if the wave travels at a constant rate, *it then follows that the time*



Figure 103 Illustration of the extensive T-shaped burn which should obstruct the hypothetical circus pathways of the flutter wave through the right auricle Aconitine was placed near the base of the auricular appendix and flutter was produced The burn was then made. As demonstrated by the motion pictures the auricle continued to flutter after the burn Electrocardiograms taken before and after the burn were identical in pattern

proximately 1 centimeter. When the motion pictures (taken at 2000 frames per second) were examined at high magnification, no contraction wave was seen to traverse the seared area, but flutter continued unchanged in the undamaged portions of the auricle.

To further demonstrate that a burn of this character effectively blocks auricular impulses, the left auricular appendix in three dogs was seared in a similar manner and auricular premature systoles were produced mechanically in the area ventral to the line of the burn. These beats were completely blocked and effectively prevented from spreading to any part of the auricles beyond the seared area. In control experiments conducted upon the same heart prior to the burn, premature systoles generated at the appendix uniformly were propagated to the rest of the heart.

The "inverted T" burn was made on the auricles during a bout of flutter As seen in Figure 103, this burn would effectively block all possible paths of an impulse travelling in a circus movement around either or both venae cavae In the motion pictures the auricles continued to flutter and, except for the areas actually burned, all parts of the auricular chambers appeared to contract simultaneously. Electrocardiograms taken before and after the burn were identical in pattern (Figure 104).

Observation 5: Flutter from a Focus at the Tip of the Right Auricular Appendix

Flutter was produced in five dogs by means of aconitine applied to the tip of the right au-

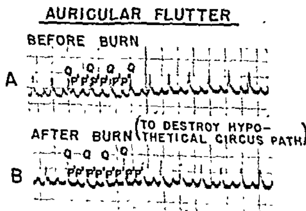


Figure 104. Electrocardiogram, lead 2, taken before and after the auricle was burned to destroy the hypothetical circus path (illustrated in Figure 103). Note that the electrocardiogram is not altered by the burn.

required for the whole journey should be approximately twice the time interval of 18 seconds cinematographic time (0.144 seconds actual time).

Lewis' experiment on the right auricle was exactly duplicated in five dogs; the onset of the second wave, as seen in the films, was timed by a stopwatch. In one animal the time of onset of the second wave was compatible with a re-entry of the original impulse if it had traveled in a circus path at a constant rate. In four animals the beginning of the second wave was considerably delayed, and its onset was as much as four times later than anticipated. In view of these results, one of two conclusions is inevitable. Either the onset of the second wave does not represent a re-entry of the original wave; or, if it does represent a re-entry, the rate of motion of the wave through the left auricle must be four times slower than through the right. The latter conclusion conflicts with Lewis' assumption that the wave traveled through both auricles at a constant rate.

Observation 3: The Contraction Wave of Flutter in Both Auricular Appendices

In Observation 1 the flutter wave was found *to progress as a broad band involving the entire width of the right auricle.* To determine whether or not this broad contraction wave continued in a circular course in the left auricle, *both* auricular appendices in four dogs were photographed simultaneously by placing the camera at a slightly greater distance from and directly over the heart. *The ectopic focus was established in the natural crevice on the caudal attachment of the interauricular septum described above; this site was essentially equidistant from corresponding points on the two auricles* (Figure 102)

If auricular flutter involved a circus movement, the left auricle should be invaded by the excitation wave subsequently to, rather than simultaneously with, the right; the left chamber should contract an appreciable time after the right. According to Lewis' data, if the circus movement were counter-clockwise, the right auricular appendix should contract 0.057 to 0.067 *second before the left; if* the movement were clockwise, the reverse would occur. On the *slow-motion picture films in which the auricular* movements are slowed 240 times, such a *difference in time of onset of the contractions* would amount to 14 seconds and could be easily detected. *No time interval between the contractions* of the two auricles was seen on the films. Most *often, the two appendices contracted* simultaneously; rarely, the left or the right appendix was found to contract *a fraction* of a second before its fellow. In the light of the known rates of conduction [383] of auricular muscle in flutter, the minute time differences occasionally recorded is far too small to be consistent with the circus movement theory.

This experiment demonstrates clearly that the contraction wave in flutter *does not pursue the circus path described by Lewis.*

Observation 4: Blocking the Path of the Flutter Wave

If the path of the contraction wave of flutter were circular, an impassable block in the course of the path should terminate the circus movement. Blocks imposed on the hypothetical circus pathway by Scherf,[541] and Brams and Katz,[64] failed to interrupt bouts of flutter. The following experiment is analogous to the work of these investigators.

In experiments in three dogs, the most effective *method for blocking an auricular* excitation wave was found to be a deep burn. A deep, *wide burn was made on* the right auricle from the superior to the inferior vena cava by carefully and repeatedly stroking the area with a red-hot glass rod until it was seared to a mottled brown and white color. The line of burn extended well upon the wall of each cava. A second burn was made perpendicular to the first and extended from it across the body of the auricle to the auriculo-ventricular groove (Figure 103). In each experiment the burn extended through the entire thickness of the auricular wall, from the *epicardial to the endo*cardial surface, the width of the burn was ap-

AURICULO-VENTRICULAR GROOVE

RIGHT AURICULAR APPENDIX

BURN

FOCUS

INFERIOR VENA CAVA

SUPERIOR VENA CAVA

SULCUS TERMINALIS

Figure 103 Illustration of the extensive T-shaped burn which should obstruct the hypothetical circus pathways of the flutter wave through the right auricle. Aconitine was placed near the base of the auricular appendix and flutter was produced. The burn was then made. As demonstrated by the motion pictures the auricle continued to flutter after the burn. Electrocardiograms taken before and after the burn were identical in pattern.

proximately 1 centimeter. When the motion pictures (taken at 2000 frames per second) were examined at high magnification, no contraction wave was seen to traverse the seared area, but flutter continued unchanged in the undamaged portions of the auricle.

To further demonstrate that a burn of this character effectively blocks auricular impulses, the left auricular appendix in three dogs was seared in a similar manner and auricular premature systoles were produced mechanically in the area ventral to the line of the burn. These beats were completely blocked and effectively prevented from spreading to any part of the auricles beyond the seared area. In control experiments conducted upon the same heart prior to the burn, premature systoles generated at the appendix uniformly were propagated to the rest of the heart.

The "inverted T" burn was made on the auricles during a bout of flutter. As seen in Figure 103, this burn would effectively block all possible paths of an impulse travelling in a circus movement around either or both venae cavae. In the motion pictures the auricles continued to flutter and, except for the areas actually burned, all parts of the auricular chambers appeared to contract simultaneously. Electrocardiograms taken before and after the burn were identical in pattern (Figure 104).

Observation 5: Flutter from a Focus at the Tip of the Right Auricular Appendix

Flutter was produced in five dogs by means of aconitine applied to the tip of the right au-

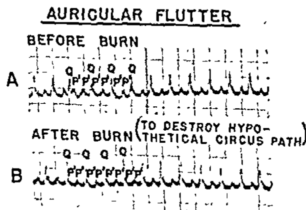
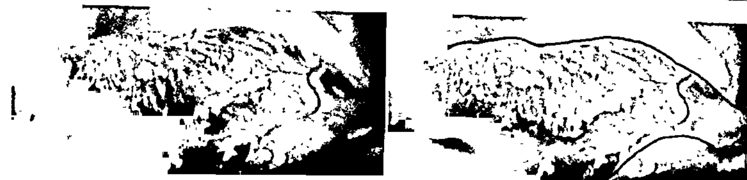


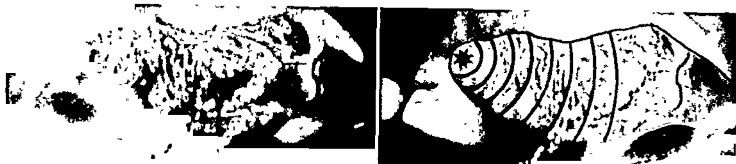
Figure 104 Electrocardiogram, lead 2, taken before and after the auricle was burned to destroy the hypothetical circus path (illustrated in Figure 103). Note that the electrocardiogram is not altered by the burn.



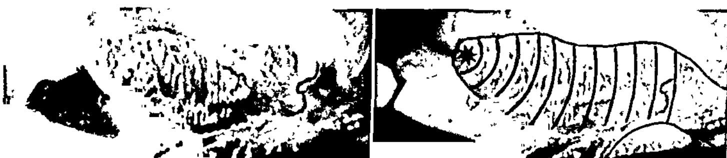
A Full diastole.



B. Flutter was produced by applying aconitine at the focus * Note contraction wave travels toward the inferior vena cava.

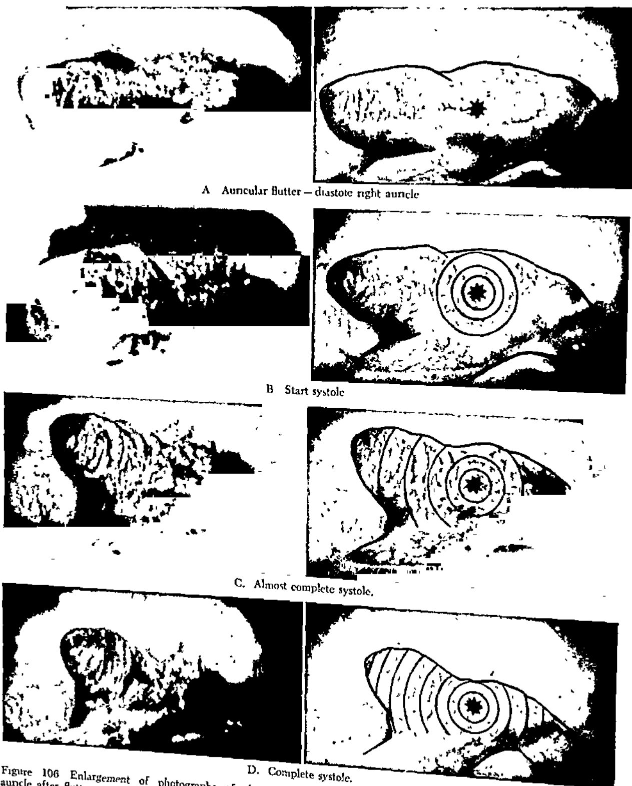


C. Mid-systole — the contraction wave has spread half way through the auricle toward the inferior vena cava



D The auricle is now in complete systole

Figure 105 Auricular flutter produced by applying aconitine to the right auricular appendix Enlarged photographs *demonstrate that the contraction wave starts at the* focus at the tip of the right appendix and travels across the auricle in a cephalocaudal direction, involves the appendix and body, and ends near the inferior vena cava This demonstrates that a circus movement is impossible because the ectopic focus at the tip of the appendix was obviously far removed from Lewis's circus pathway. Furthermore, no return wave can be seen in the cinematographs going in the opposite direction in the appendix, as would have to be present to complete the circus pathway.



A Auricular flutter — diastole right auricle

B Start systole

C. Almost complete systole.

D. Complete systole.

Figure 106 Enlargement of photographs of the right auricle after flutter was produced by aconitine placed near the center of the right auricle. It can be seen that the con- traction

ricular appendix (Figure 105). Cinematographs demonstrated that the contraction waves started at the site of application and traveled across the auricle in a cephalocaudal direction, involving successively the appendix and body, and ended near the inferior vena cava. The relaxation waves pursued an identical course. When the aconitine focus was cooled or amputated, flutter immediately ceased.

Observations on the flutter wave initiated at this particular site proved that a circus *movement was impossible for the following reasons:* (1) the ectopic focus on the tip of the appendix obviously was far removed from the pathway hypothesized by Lewis, yet when the focus was excised, the arrhythmia abruptly terminated; hence, no independent circus movement could exist. Furthermore, the focus could not be located in the region of a daughter wave, for in that case again, abolition of the focus should not affect a main circus pathway and therefore would not terminate the arrhythmia. (2) If the ectopic focus at the tip of the appendix initiated a circus movement, the wave would have to return to the focus to complete the circuit and the appendix should then contract from base to apex immediately before the onset of the succeeding systole. No such contraction was apparent on the films. The wave left the ectopic focus and, as it traveled in a cephalocaudal direction, involved successively the appendix and body of the auricle. The contraction wave was followed by a relaxation wave which pursued the same course. Each succeeding systole was initiated and propagated in an identical manner, nothing was seen resembling a wave returning from the body through the appendix *to the initiating focus.**

This observation is consistent with Observations 1, 2, 3 and 4: Circus movement, as formulated by Lewis, cannot be the mechanism of auricular flutter.

* The suggestion has been made that the circus path embraced the natural obstacles in the auricles (i e, the two auriculo-ventricular rings) Obviously, movement of a wave along such a path is incompatible with the results of observation 4

Observation 6: Course of the Contraction Wave of Flutter from a Focus at the Center of the Right Auricle

The preceding five observations furnished conclusive evidence refuting the circus movement theory. *The exact nature of the flutter* wave, however, was not yet clearly demonstrated. For this purpose, flutter was produced from an ectopic focus in the *center* of the right auricle. The resulting wave could be recorded cinematographically from the moment of its inception until it reached its ultimate destination.

A focus in the center of the right auricle was created by application of the aconitine mixed with India ink and the presence of flutter confirmed by electrocardiograms. Motion pictures of the auricle were taken at 2000 frames per *second. The contraction wave initiated at the* aconitine focus and radiated from it in all directions simultaneously (Figure 106). Diastole started at the aconitine focus and followed the same course as systole. These results were uniform in 10 consecutive experiments. Identical observations were made in similar experiments *in which flutter was produced by post-electrical* stimulation.

The results of this experiment are of fundamental *significance; for the first time a flutter* wave was actually visualized throughout its en*tire course. The wave arises from an ectopic* focus and spreads in all directions simultaneously; it does not follow the unidirectional course hypothesized in the circus movement theory.

DISCUSSION

It has been clearly demonstrated by the cinematographic observations herein set forth, that auricular flutter consists of rapidly repeating contraction waves arising from an ectopic focus from which the waves spread through the auricles in all directions simultaneously. There is no circus movement.

The validity of the circus movement theory first came into question when it became appar-

ent on the films that the contraction wave in flutter did not pursue a narrow band, did not emit daughter waves, and did not travel in a circular path. Further cinematographic observations revealed that the contraction wave in flutter originated in a single ectopic focus and spread through both auricles in all directions simultaneously. It has been pointed out that Lewis' data were incomplete; he himself recognized the importance of the gap in his determinations which resulted from his inability to trace the course of the excitation wave over the body of the left auricle.

In an attempt to reconcile our observations with Lewis' data, we were able to confirm cinematographically his finding (Figure 97) that the contraction wave of flutter arising in the region of the inferior vena cava spreads cephalad in the right auricle. It is his assumption that the wave turns and continues in a unidirectional course through the body of the left auricle back to the point of stimulation that we found unjustified. From our cinematographically recorded experiments, we are forced to conclude that the flutter wave spreads throughout both auricles simultaneously. Since the body of the left auricle is immobile, however, the cinematographs do not reveal the course of the contraction wave in this area. In order to complete our evidence on the course of the flutter impulse, it became necessary to explore the body of the left auricle electrocardiographically. The results of this investigation are reported in the following chapter.

It is interesting to note that MacWilliam,[122] the first to introduce the term "flutter," as far back as 1887 suggested that the disturbance was due to rapid, repetitive, impulses from an ectopic focus. More than 60 years later, our observations confirm his theory

SUMMARY AND CONCLUSION

The evidence for and against Lewis' circus movement theory of the mechanism of auricular flutter has been re-examined. Because of incomplete exposure, the body of the left auricle has not heretofore been studied; Lewis' evidence was, therefore, incomplete.

In the present study, both the left and right auricles were completely exposed in 40 dogs. Auricular flutter was produced experimentally by post-electrical stimulation or, in most instances, by aconitine. Cinematographically, the wave was seen to move as a broad band; no evidence of a narrow "mother" path or of "daughter" waves was discovered. When the ectopic focus was established in the region of the caudal attachment of the interauricular septum, the auricles appeared to beat simultaneously; this phenomenon is incompatible with circus movement. After flutter was established, blocking the hypothetical circus pathway by means of a burn did not interrupt the arrhythmia. From an ectopic focus established at the tip of the right auricular appendix, the impulse spread in a cephalocaudal direction, involving successively the appendix and body. Each successive systole started in the same manner; no returning "re-entry" wave was seen. When an ectopic focus was established at the center of the right auricle, the onset and entire course of the flutter wave was visualized, the contractions and succeeding relaxations were observed to spread away from the focus in a centrifugal manner.

From these cinematographic observations, the conclusion is inevitable that flutter results from rapidly recurring impulses which spread out in all available directions simultaneously from a single ectopic focus. There is no circus movement.

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Observations on the flutter wave initiated at this particular site proved that a circus movement was *impossible for the following* reasons: (1) the ectopic focus on the tip of the appendix obviously was far removed from the pathway hypothesized by Lewis, yet when the focus was excised, the arrhythmia abruptly terminated; hence, no independent circus movement could exist. Furthermore, the focus could not be located in the region of a daughter wave, for in that case again, abolition of the focus should not affect a main circus pathway and therefore *would not terminate the arrhythmia.* (2) If the ectopic focus at the tip of the appendix initiated a circus movement, the wave would have to return to the focus to complete the circuit and the appendix should then contract from base to apex immediately before the onset of the succeeding systole. No such contraction was apparent on the films. The wave left the ectopic focus and, as it traveled in a cephalocaudal direction, involved successively the appendix and body of the auricle. The contraction wave was followed by a relaxation wave which pursued the same course. Each succeeding systole was initiated and propagated in an identical manner; nothing was seen resembling a wave returning from the body through the appendix to the initiating focus.*

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OBSERVATION 6: COURSE OF THE CONTRACTION WAVE OF FLUTTER FROM A FOCUS AT THE *CENTER OF THE RIGHT AURICLE*

The preceding five observations furnished conclusive evidence refuting the circus movement theory. The exact nature of the flutter wave, however, was not yet clearly demonstrated. For this purpose, flutter was produced *from an ectopic focus in the center* of the right auricle. The resulting wave could be recorded cinematographically from the moment of its inception until it reached its ultimate destination.

A focus in the center of the right auricle was created by application of the aconitine mixed with India ink and the presence of flutter confirmed by electrocardiograms. Motion pictures of the auricle were taken at 2000 frames per second. The contraction wave initiated at the aconitine focus and radiated from it in all directions simultaneously (Figure 106). Diastole started at the aconitine focus and followed the same course as systole. These results were uniform in 10 consecutive experiments. Identical observations were made in similar experiments *in which flutter was produced by post-electrical* stimulation.

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From these cinematographic observations, the conclusion is inevitable that flutter results from rapidly recurring impulses which spread out in all available directions simultaneously from a single ectopic focus. There is no circus movement.

CHAPTER VI

Electrocardiographic Studies of Auricular Flutter

PRIOR to our cinematographic studies of auricular flutter, all experimental work on this arrhythmia and all inferences concerning its mechanism were based on electrocardiographic, sphygmographic, polygraphic and kymographic investigation. In order to correlate the cinematographic observations with previous research in this field, Lewis' original experiments were duplicated and extended in our laboratory. In addition, electrocardiographic studies were made on the body of the left auricle and the interauricular septum, regions heretofore unexplored. Each type of experiment was repeated five to eight times, with uniform results.

In the present study, the course of the excitation wave of auricular flutter was traced through the right and left auricles and the interauricular septum. Paired electrodes were placed at strategic sites on the auricles; simultaneous tracings from these electrodes were recorded by a direct-writing multiple-channel electrocardiograph (Appendix). Determinations of the course and speed of the flutter excitation wave were based on two fundamental principles of electrocardiography: (1) Since the intrinsic deflection in a tracing is recorded as the impulse passes beneath the electrode, the differences in time of arrival of the flutter impulse at the simultaneously recording electrodes could be calculated (2) The shape of the auricular deflections varies with the direction of the impulse in relation to the electrodes, hence, changes in configuration of the waves in the tracings revealed the direction of the flutter impulse at different sites on the auricles.

Lewis' circus movement theory was based upon observations of electrically induced flutter in the dog; he and his associates attempted to trace the course of the flutter excitation wave by means of paired electrodes placed on various parts of the right auricular body and the right and left auricular appendices. As shown in Figure 97, the values obtained in the right auricle by Lewis indicate that the flutter wave traveled from right to left, from 0.0000 to 0.0800 second across the length of the right auricular wall. That the wave actually followed this path through the right auricle is unquestioned; the electrocardiographic determinations were duplicated in this laboratory and confirmed by cinematographic observation. The supposition that the impulse turns sharply around the superior vena cava and descends through the left auricle to (point 0.0900), as postulated by Lewis, is not proved by this data.[364, 385]

TIMING THE INTRINSIC DEFLECTIONS IN DIRECT AURICULAR LEAD ELECTROCARDIOGRAMS OF AURICULAR FLUTTER

OBSERVATION 1. COURSE OF THE FLUTTER EXCITATION WAVE IN THE RIGHT AURICLE

This experiment was designed to trace electrocardiographically the course of the excitation wave of auricular flutter and to verify the cinematographic observation that a flutter wave originating at an aconitine focus in the center of the auricle does not follow a unidirectional path but spreads in all directions simultaneously (Chapter V, Observation 6).

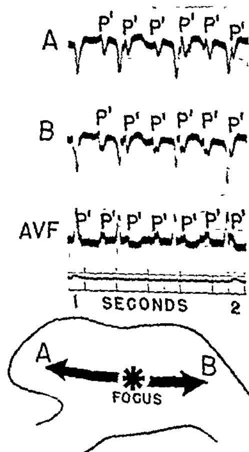


Figure 107 Electrocardiograms of flutter established by placing aconitine at *the center of the right auricle*, electrodes A and B are on each side of the focus and equidistant (approximately 2 cm) from it The intrinsic deflection recorded from each electrode occurs at almost the same instant Therefore, the impulse must travel from the focus to A and B simultaneously

An aconitine focus (F) was produced in the center of the right auricle (Figure 107). Electrodes A and B were placed approximately 2 centimeters from the *focus*, one cephalad and the other caudad Auricular flutter was then produced in nine dogs and electrocardiograms recorded simultaneously *from each* electrode. As seen in Figure 107, the corresponding intrinsic deflections derived *from the* two electrodes were inscribed at *almost exactly* the same instant (within 0 005 second), hence, the impulse must have *traveled in both directions simultaneously* If the impulse had pursued a unidirectional course, it would have reached one electrode before the other — A earlier than B if the course were counter-clockwise, B earlier than A if the movement were clockwise. Identical results were obtained when flutter was produced by electrical stimulation and persisted as a post-stimulatory effect for a period sufficient to permit study.

This experiment demonstrates that the course of the flutter excitation wave from the center of the right auricle is consistent with the course of the corresponding contraction wave seen in cinematographic studies. Rapidly recurring impulses starting from an ectopic focus in the center of the right auricle do not spread along a unidirectional pathway but, as shown by this experiment, spread in opposite directions.

Observation 2: Course of the Flutter Excitation Wave in Both Auricles

Because the body of the left auricle is essentially immobile, no direct information concerning the course of the flutter contraction wave in that structure could be derived from the cinematographs. On the basis of electrocardiographic Observation 1 of this Chapter and cinematographic Observation 6 (Chapter V), both of which demonstrate that the *wave spreads* away *from* its site of origin in more than one direction simultaneously, it would be expected *that when* Lewis' experiment was duplicated, the course *of* the flutter wave through the body of the left auricle *might* be *in* a direction *opposite* to that postulated by him. In the following experiments the course *of the flutter* excitation wave was *traced* through both auricles in 15 dogs.

Simultaneous Activation of Both Auricles: Auricular *flutter* was produced by creating an aconitine focus on the caudal attachment of the interauricular septum at a point between the inferior vena cava *and* the pulmonary vein from the lower lobe of the left lung. Electrodes A and B were placed equidistant from the focus on the bodies of the *right* and left auricles respectively (Figure 108). Electrocardiograms recorded simultaneously from the two electrodes reveal that the flutter *impulse* reached points A and B at the same instant (Figure

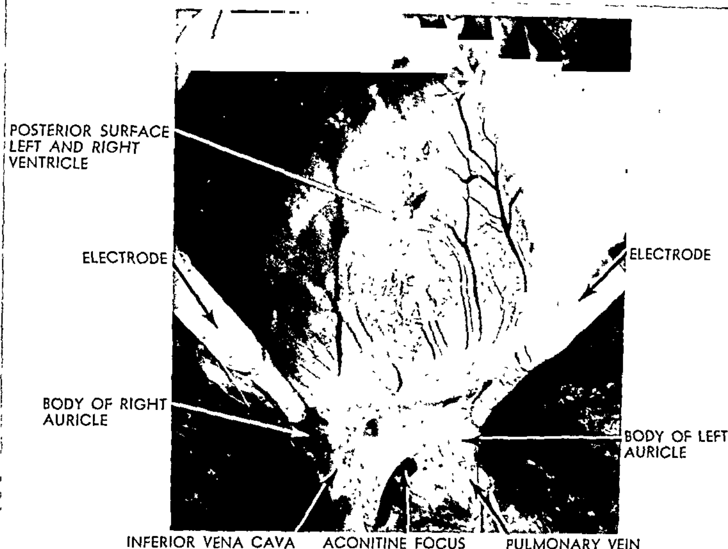


Figure 108. Photograph of the heart elevated so that the apex is cephalad to demonstrate the relationship of the *posterior surface of the heart, site of aconitine application and* placement of electrodes. Aconitine was applied to the natural crevice formed by the inferior vena cava and a branch of the pulmonary vein In this experiment (observation 2) electrodes were placed on each auricle equidistant from the aconitine focus

109). Apparently the impulse, after leaving the aconitine focus, spread over both auricles simultaneously; had the flutter wave traveled in a unidirectional circular path, a considerable period of time would have elapsed between activation *of the two auricles. These electrocardiographic* findings are analogous to the cinematographic observation that the right and left auricular appendages contract simultaneously during flutter (Chapter V).

Direction of the Flutter Wave in Each Auricle: Flutter was produced from an aconitine focus between the two auricles at the same site as in experiment 2A (Figure 108). Paired unipolar electrodes were placed on the body of each auricle; simultaneous electrocardiograms were taken from each pair of electrodes successively. (In each instance A refers to the electrode nearest the focus, and B to the electrode farthest from the focus.) Figures 110 and 111 show that in both auricles the intrinsic *deflections from electrode A were recorded be*tween 0.03 and 0.04 second before the corresponding deflections from electrode B, indicating that the flutter impulse reached point A before point B in each instance. It was thus demonstrated that the flutter impulse traveled from the aconitine focus across both auricles in approximately the same direction simultaneously.

For the first time the body of the left auricle has been explored electrocardiographically, and

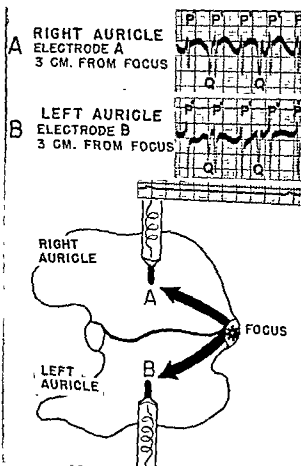


Figure 109 Electrocardiograms of flutter established by placing aconitine at the caudal attachment of the interauricular septum Electrode A is on the right auricle 3 cm from the focus, electrode B is on the left auricle 3 cm from focus The onset of the intrinsic deflection occurs simultaneously in each auricle If there were a circus movement, the intrinsic deflection from one electrode should be recorded a significant interval before that from the other because the impulse would have to travel first up one auricle and then down the other.

the course of the flutter wave through both auricles has been charted. *The flutter wave apparently spreads from the focus of origin in different directions simultaneously, it does not pursue a circus pathway*

Observation 3. The Interauricular Septum in Flutter

The concept of circus movement embraces the supposition that the excitation wave of flutter circulates in one direction only along a narrow path in a *single* plane For the impulse to remain indefinitely within the delimited path and to continue to circulate in one direction only, it would be necessary for the path to be surrounded by refractory tissue on all its borders except in its forward direction. The pathway postulated by Lewis would cross the septum twice during each circuit, once near the superior vena cava and again just caudad to the inferior vena cava. For the circus movement to exist, the septum also would have to be refractory. Otherwise, as the excitation wave passed over the septum it would be transmitted through this structure to adjacent parts of the auricle, the impulse would be short-circuited, and the arrhythmia would terminate. These considerations render circus movement impossible unless one makes the highly improbable assumption that the hypothetical circus path is surrounded on all sides, *including the septum*, by refractive tissue which effectively insulates the path and prevents the impulse from spreading to any other portion of the auricles.*

As demonstrated in Chapter I, the interauricular septum and the two auricles have direct muscular and endocardial continuity; they may therefore be expected to react as a unit in response to effective stimuli. It also was demonstrated that during sinus rhythm the septum contracts and conducts electrical impulses in the same manner as do other portions of the auricles. In order to determine whether or not the septum is activated in flutter, the following experiments were performed in four dogs.

Conductivity of the Interauricular Septum in Flutter: A purse-string suture was placed around the tip of the right auricular appendix. A small incision was made distal to the suture, a non-polarizable electrode was immediately introduced and the suture tightened around the electrode. The tip of the electrode was then directed against the interauricular septum and held in place manually. Flutter was produced by application of aconitine to the surface of the body of the right auricle. During the experiment manual palpation through the right auric-

* If this were true, the "daughter waves" postulated by Lewis could not occur since no impulse could penetrate the mantle of refractory tissue.

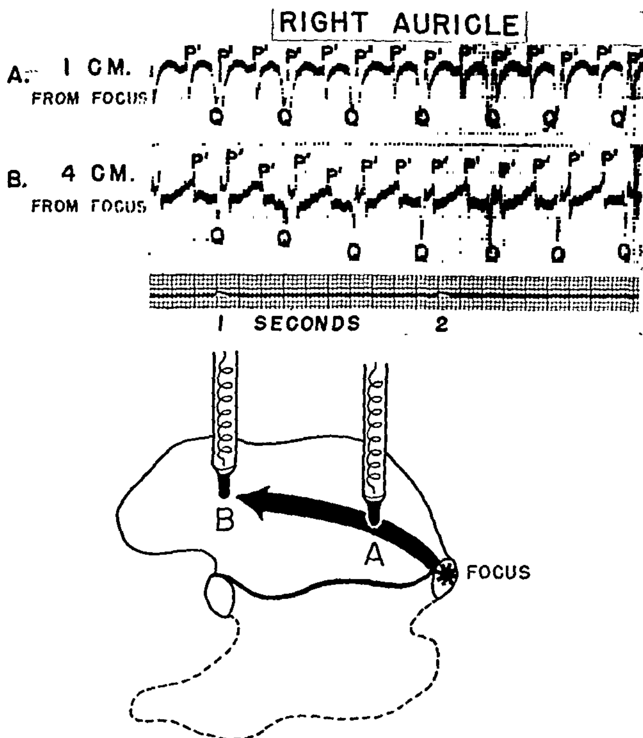
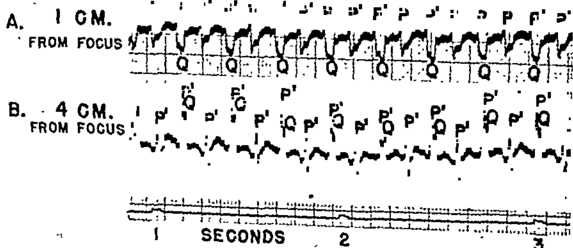


Figure 110 Electrocardiograms of flutter established by placing aconitine (focus) at the caudal attachment of the interauricular septum Both electrodes are on the right auricle, A is 1 cm from the focus, B is approximately 4 cm from the focus The onset of the intrinsic deflection from A is about 0 03 second ahead of that from B Therefore, the impulse must travel from A to B.



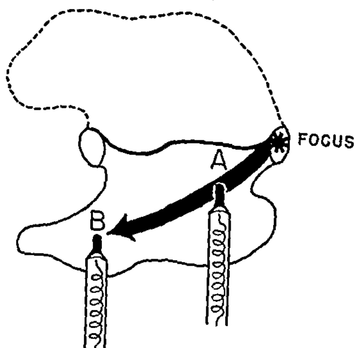


Figure 111 The same experiment as in Figure 110, except that both electrodes are now on the left auricle Electrode A is 1 cm from the focus, electrode B is approximately 4 cm from the focus The onset of the intrinsic deflection from A is about 0.01 second before that from B Therefore, the impulse must travel from A to B. If there were a circus movement, it would have to go from B to A

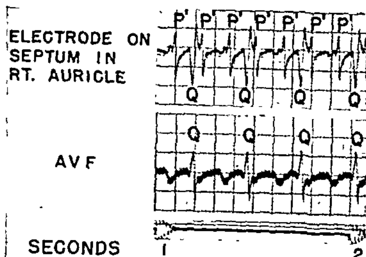


Figure 112. Simultaneous electrocardiograms from a dog with 2:1 auricular flutter, one record (above) is from an electrode on the septum and the other is lead AVF (below). The septum is obviously activated during flutter and the P′ waves are of similar configuration to those found on the walls of the right and left auricles

ular wall revealed that the tip of the electrode was against the septum. At the termination of the experiment, the auricle was opened and the electrode was again found in contact with *the septal musculature.* Continuous electrocardiograms recorded from the intra-auricular electrode presented the appearance characteristic of auricular flutter (Figure 112) The contour of the auricular complexes coincided in every way with those recorded in direct leads from the surface of the auricle.

Thus, in auricular flutter, as in normal sinus rhythm, the interauricular septum conducts the cardiac impulse in the same manner as does any other part of the auricle. This observation *is contradictory to the circus movement theory* since the septum would necessarily short-circuit the impulse to other parts of the auricles On the other hand, the observation is compatible with the authors' concept that the flutter impulse originates in an ectopic focus and spreads outward from the focus to invade all portions of the auricles.

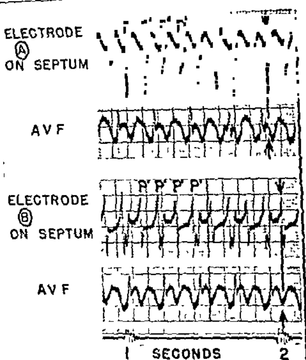
Course of the Flutter Impulse in the Interauricular Septum: Flutter was produced by aconitine placed in the crevice at the caudal attachment of the inter-auricular septum in four dogs. A non-polarizable electrode was introduced *into the right auricle* through an incision in the right auricular appendix, as described above, and placed in contact with the septum *just ventral and cephalad to the tricuspid* valve in an area near the aconitine focus; *electrocardiograms* were recorded from this electrode. During the same episode of flutter, the electrode was moved approximately 3 centimeters cephalad on the septum and additional records were made. The time of arrival of the impulse at these two points was determined with a simultaneously recorded lead AVF.

The intrinsic *deflections recorded from point* A near the aconitine focus were inscribed before *those from point B which* was more distant from the focus (Figure 113). Hence, the course of the impulse in the septum was the same as that observed in other parts of the auricles; the impulse originated in the ectopic focus and spread away from the focus.

SHAPE OF THE AURICULAR DEFLECTIONS IN DIRECT LEAD ELECTROCARDIOGRAMS OF AURICULAR FLUTTER

As noted in Chapter I, *the shapes of the deflections* recorded from direct auricular leads are of fundamental importance to the study of the excitation wave of normal sinus rhythm. These relationships apply also in auricular flutter. An electrode placed at or near the site of origin of an auricular impulse will register primarily a negative wave of depolarization. As the electrode is moved away from the focus, an *initially positive* wave *appears,* followed by a negative deflection. As the distance from the *site of origin of the impulse* increases, the positive deflection progressively grows larger in amplitude and *duration while the negative* deflection eventually disappears. When the direction of the impulse is toward the electrode, a positive deflection is recorded, if the direction is away from the electrode, the *deflection* is negative.

If there were a *pure, self-perpetuating circus* movement traveling in an *identical manner* in all parts of a circus pathway, all the deflections on the main pathway would have the same



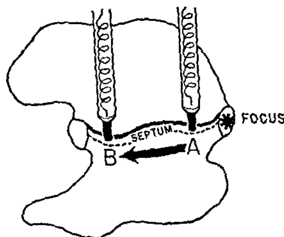
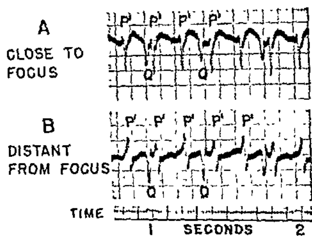


Figure 113 Direct auricular lead electrocardiograms of flutter from a focus established at the caudal attachment of the interauricular *septum*, recorded simultaneously with lead AVF as a reference lead Two electrodes are on the septum, A is closer to the focus than is B It is seen that the intrinsic deflection was recorded first from electrode A and then from electrode B Therefore, the impulse must have traveled through the septum in a caudocephalic direction from the focus

configuration since such a pathway would be without beginning or end. It can be stated, however, that this is not the case It was repeatedly observed in flutter that the configurations of the deflections from direct electrodes varied with their distance from the focus. Similar variations are readily noted in Lewis' own records made from direct auricular leads. The following experiment illustrates these variations.

Observation 4: Variations in Configuration of the Auricular Deflections in Direct Lead Electrocardiograms of Auricular Flutter

Focus Between the Left and Right Auricles: In each of five dogs, auricular flutter was in-



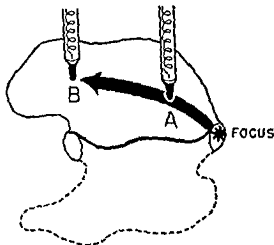


Figure 114. Electrocardiograms of flutter from an aconitine focus established at the caudal attachment of the interauricular septum Electrode A is close to the focus and electrode B is distal to the focus The record from A shows that the auricular deflection is essentially a pure negative wave. The record from B shows a large positive wave preceding the negative deflection The intrinsic deflection from A is recorded about 0.02 second earlier than from B Therefore, from both the configurations of the waves and the onset of the intrinsic deflections, the impulse must have traveled from A to B.

itiated by aconitine placed at the junction of the inferior vena cava and the pulmonary vein. One electrode (A) was placed adjacent to the focus on the body of the right auricle and another (B) distant from the focus on the right auricular appendix. As illustrated in Figure 114, the deflection registered at electrode A was mainly negative while that at electrode B exhibited a large positive component.

The above findings indicate that the impulse at electrode B was a considerable distance from its origin. While this disproves the presence of a self-perpetuating impulse, it does not rule out the possibility of a circus movement which arises anew from the same focus with each cycle. In such an instance the distance between the impulse and the focus would gradually increase as the circuit neared completion and the auricular deflections would grow progressively more positive as the site of origin was approached on the return journey. When experiment 4 was repeated with paired direct electrodes placed in the body of the left auricle, the auricular deflections were predominately negative in the tracing from the electrode nearest the focus. This result indicated that, as in the right auricle, the course of the impulse over the body of the left auricle was *away* from the focus. It was thus demonstrated that the flutter impulse originated at the ectopic focus and spread outward from the focus across both auricles in different directions simultaneously.

Figure 113 shows that the same variations in the shape of the auricular deflections with reference to the distance from the focus occur when the recording electrodes are placed on the interauricular septum. When the electrodes are equidistant from the focus on any part of the auricle, the configuration of the auricular deflections recorded from each is similar (Figures 107 and 109).

Focus on Tip of Right Auricular Appendix: Auricular flutter was produced by applying aconitine to the tip of the right auricular appendix. One electrode was placed on the right auricular appendix adjacent to the focus, and another at a distant point on the body of the auricle. Figure 115 shows that the deflection from electrode A was primarily negative, while that from electrode B was mainly positive, indicating again that the impulse spread *away* from the focus.

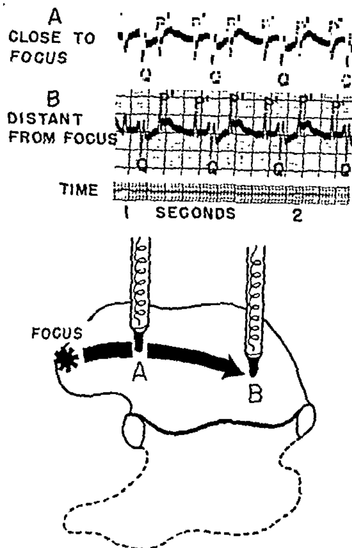


Figure 115. Electrocardiograms of auricular flutter established by placing aconitine on the tip of the right auricular appendix. As in Figure 114 the deflection from electrode A (nearer the focus than B) is essentially a negative deflection. The deflection from B is essentially a positive deflection. The time of onset of the intrinsic deflection in A is 0.03 second earlier than in B. Therefore, the impulse must originate in the appendix and travel in a cephalo-caudal direction

SUMMARY AND CONCLUSIONS

The cinematographic observations of auricular flutter described in the previous chapter have been correlated with electrocardiographic investigations in which the course of the excitation wave was traced by means of direct auricular leads. Two methods of study were

used: (1) determination of the relative times of arrival of the impulse at strategic areas on various parts of the auricles, including the interauricular septum and the body of the left auricle, regions heretofore unexplored; and (2) observation of the variations in the shape of the auricular deflections in relation to the distance of the recording electrodes from the focus.

(1) Whether an aconitine focus was established on the right auricle, on the left auricle, or between the two auricles on the caudal border of the interauricular septum, simultaneously recorded direct lead electrocardiograms revealed that the flutter impulse reached electrodes equidistant from the focus at the same time; when the electrodes were placed at unequal distances from the focus, the impulse arrived at the more distant electrodes later than at the less distant electrodes (2) In auricular flutter from an aconitine focus between the two auricles on the caudal attachment of the interauricular septum, auricular deflections were derived from electrodes placed at varying distances from the focus, the shapes of these deflections were studied in relation to their distance from the focus Whether the electrodes were placed on the right auricle, the left auricle or the interauricular septum, consistently the auricular complexes from electrodes near the focus were predominantly negative, as the distance between the electrode and the focus increased, the complexes became more positive Both methods demonstrated that the course of the flutter excitation wave was identical in the right auricle, in the left auricle and in the interauricular septum, it traveled outward from the focus at the same speed in all directions simultaneously

Thus the course of the excitation wave of auricular flutter is identical with the course of the corresponding contraction wave as demonstrated cinematographically (Chapter V).

The relationship of both the timing and the configurations of the auricular deflections to the distances of the recording electrodes from the focus is identical in normal sinus rhythm, premature auricular systole, paroxysmal auricular tachycardia and auricular flutter. The variations are the same in the right and left auricles, in the two appendices and in the interauricular septum If the basic electrocardiographic concept concerning the propagation of the cardiac impulse is correct, in each of these arrhythmias the impulse radiates outward from the focus *in all available directions*. The flutter wave does not pursue the unidirectional course hypothesized by Lewis.

Because the body of the left auricle is immobile upon cinematographic observations and heretofore has not been subjected to direct electrocardiographic study, the *entire course of* the flutter wave through both auricles has never been demonstrated. Lewis himself admitted that the evidence supporting his circus movement theory was inconclusive because the portion of the circus pathway lying within the *body of the left auricle* was merely inferred. In the present study, the experiment from which Lewis derived his theory was duplicated while electrocardiograms were recorded from electrodes placed on the exposed body of the *left auricle*. The excitation wave of auricular flutter was thus shown to travel through the left auricle in a direction opposite that of the hypothetical circus movement.

It was demonstrated that the interauricular septum is anatomically and physiologically an integral part of the auricles, and that it conducts the flutter impulse in exactly the same manner as other portions of the auricles. Because of this unity of structure and function, the septum would transmit *any impulse* arriving along its borders. This is inconsistent with the existence of an unidirectional circus movement, since the impulse would be short-circuited whenever it crossed the interauricular septum.

On the basis of the experimental observations reported in this and the preceding chapter, two conclusions are inevitable:

1. Auricular flutter consists of rapidly recurring impulses arising from an ectopic focus and spreading in *all available directions simultaneously*. The circus movement theory advanced by Lewis is invalid.

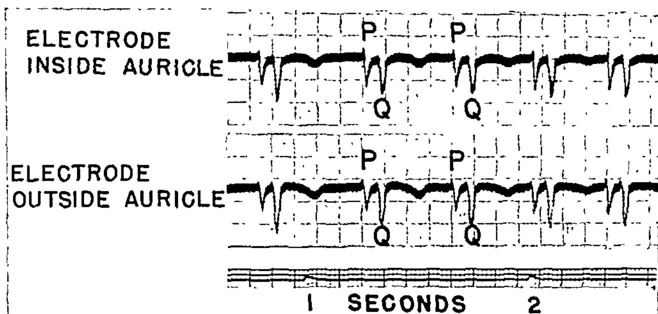


Figure 116 Electrocardiograms taken simultaneously with two electrodes. The auricular complexes from *the electrode on the endocardial surface of the* auricle are the same as the auricular complexes from the corresponding pericardial surface of the auricle The similarity of the endocardial and pericardial complexes of the auricles is found in all parts of the auricles providing the paired electrodes are exactly opposite each other

2. The contraction and excitation waves of auricular flutter are identical in course to each other and to the corresponding waves of auricular premature systoles and auricular paroxysmal tachycardia.

COLLATERAL OBSERVATIONS

Auricular Intrinsic Deflections from Epicardial and Endocardial Surfaces

It has been maintained that the deflections derived from the endocardial surface of the auricles differ from those obtained from the corresponding epicardial surface. *In this respect*, the electrocardiographic response of the auricules was thought to be analogous to that of the ventricles. During the course of our experimental studies, many electrocardiograms were recorded from paired electrodes placed on exactly corresponding endocardial and epicardial surfaces of the auricles. Regardless of the part of the auricles studied, the configurations of the deflections recorded simultaneously from corresponding epicardial and endocardial surfaces always resembled each other and in many instances were identical (Figure 116). This was true during *sinus rhythm*, auricular premature systoles, auricular paroxysmal tachycardia, and auricular flutter.

Although the auricular wall in the dog admittedly is thin, it still possesses measurable *anatomic thickness. The above observations*, however, seem to indicate that this thickness is electrocardiographically insignificant and the auricular wall reacts as a surface area only (two dimensional). The ventricular wall, being much thicker, responds in the manner expected of a three dimensional structure

Variation in Mechanical Response Principle of Dissociation

Cinematographically, in normal sinus rhythm and *in slower-rate tachycardias produced by* electrical, chemical or mechanical stimulation, the mechanical response of the auricular muscle is the same during every beat; each systole and each diastole appears identical with its predecessor. As the auricular rate approaches 300 beats per minute, distinct variations in the auricular contractions become apparent Although regular in rhythm, the contractions become irregular in force, some are strong, others are weak, some are almost invisible. This phenomenon was repeatedly observed in a large

number of experiments, of which the following is illustrative.

Tachycardia was produced in a dog by electrical stimulation at a rate of 220 impulses per minute When observed cinematographically, all auricular contractions were evenly spaced and completely regular in force, each systole was identical with the others, and each was followed by a ventricular response. When the rate of stimulation was increased to 280 impulses per minute, the auricular contractions became irregular in force; the rhythm, however, was still regular, and a ventricular response followed each auricular contraction When the auricles were driven at rates over 300 impulses per minute, some of the stimuli failed to evoke a perceptible mechanical response.

In such experiments, simultaneous electrocardiograms demonstrated that the weak or indistinct auricular contractions, and even those which were completely invisible on the motion picture films, inscribed depolarization waves of the same shape and voltage as those associated with the strongest beats This manifestation of the principle of dissociation – electrical activity in the absence of apparent mechanical activity – has been observed in our laboratory in many experiments (in excess of 25). The reasons for such lack of correlation between mechanical muscular contractions and simultaneous electrical activity is not known.

As might be expected, the contraction waves of auricular flutter display variations in force similar to those observed in auricular paroxysmal tachycardia, because the rate of flutter is more rapid, these changes are correspondingly more pronounced As in tachycardia, the variations in mechanical activity are not accompanied by similar variations in the auricular deflections in the electrocardiogram.

Because of the frequency of mechanical contractions when the auricle is driven at a rapid rate, it usually is impossible to distinguish between the high-rate arrhythmias with the unaided eye: they all resemble "fibrillation." The differentiation is further complicated by the "double contraction" phenomenon which may occur either during normal sinus rhythm or during arrhythmias (Chapter I). On occasion, clinicians have attempted to diagnose high-rate arrhythmias in humans by observing the exposed heart during surgical operations.[121] Lewis[351] thought he could recognize specific arrhythmias in the exposed heart of the horse without the aid of electrocardiograms. The foregoing discussion suggests that great caution must be exercised in making such diagnoses. Clinicians have long known that the auricular sounds in flutter are variable; electrocardiograms recorded during these phonetic variations are unchanged (Figures 95 and 96, Chapter V.)[340] Phonocardiograms made during regular sinus rhythm with complete auriculo-ventricular dissociation do not show variations in *sound* waves. This clinical evidence suggests that changes in mechanical response without alterations in the electrocardiogram occur during the high-rate auricular arrhythmias in man as well as in experimental animals.

CHAPTER VII

An Electrocardiographic Analysis of the Structure of the Auricular Deflections of Auricular Flutter and Tachycardia

INTENSIVE study has shown that the ventricular complex consists essentially of waves of depolarization and repolarization. The auricular complex, unlike the ventricular deflection, has received little attention from electrocardiographers and the nature of its component parts is not as well understood. This is especially true in tachycardia and flutter, where the basic phenomena of auricular depolarization and repolarization have been completely ignored.

Auricular flutter is usually characterized by a specific undulating wave (continuous undulation of the base line) generally believed to represent transmission of the excitation wave in a circus pathway around the venae cavae. This configuration, considered specific for the arrhythmia, is held to be unrelated to any other type of auricular complex. The saw-tooth undulations of flutter are designated as "F" waves, implying that the flutter waves are produced by a mechanism different from that of the P waves of sinus rhythm and the P' waves of auricular premature systoles and auricular paroxysmal tachycardia. Evidence derived from a study of the momentary atrial axis is thought to support the circus movement concept of flutter.

The familiar continuous undulations of the flutter waves apparently have induced clinicians and electrocardiographers to accept the circus movement theory of flutter This is understandable for such undulations might well be interpreted as a graphic representation of waves traveling in a circular pathway. If the flutter waves did not exhibit an undulating appearance, it is doubtful that the circus movement theory would have achieved general acceptance, especially in view of the admittedly incomplete supporting evidence.

The normal P wave represents depolarization of the auricular musculature and is referred to as the excitation wave (Chapter I); it is sometimes followed by a Ta segment which represents auricular repolarization. In the normal electrocardiogram the Ta segment generally is insignificant or masked by the ventricular complex. In the presence of auriculo-ventricular block, if no ventricular complex interferes, a small Ta wave can be clearly seen in the limb leads in about 50 per cent of cases; in the other 50 per cent it is absent.

The electrocardiographic evidence presented in this chapter will show that the general principle of depolarization and repolarization applies also to flutter and tachycardia. The "F" wave of flutter is not a unique wave unrelated to other electrocardiographic deflections, but is probably composed of two components, namely, a wave of depolarization followed by a wave of repolarization. This undulating type of auricular complex representing the same phenomena may also occur in auricular paroxysmal tachycardia. The wave of depolarization will be considered in Part I of the chapter and the wave of repolarization in Part II.

Part I

DEMONSTRATION OF THE WAVE OF DEPOLARIZATION IN FLUTTER AND TACHYCARDIA

Experiment 1A. Demonstration of the Excitation Wave in the Flutter Complex by Means of Direct Auricular Leads in Dogs

This experiment identifies and delineates the approximate onset and termination of the wave of depolarization in the flutter undulation.

Auricular flutter was initiated by aconitine placed at the extreme caudal end of the auricle over the interauricular septum. One unipolar electrode was placed on the auricle close to the focus and another was placed on the tip of the appendix of the same auricle at a site as far removed from the first electrode as possible. Simultaneous recordings were made from these two direct leads and lead AVF (Figures 117 and 118). By projecting the tracings from the direct auricular leads on the simultaneously recorded tracing from the limb leads the excitation wave in the limb lead can be approximately delineated. The start of the excitation wave, as indicated by the intrinsic deflection from the electrode nearest the aconitine focus, is at the beginning of the descending limb of the trough in the flutter complex of the limb lead. The termination of the excitation wave is usually at the junction of the ascending limb with the upward bowing of the flutter complex.

Similar results were obtained when electrodes were placed directly on the septum (Figure 119). Flutter was produced by application of aconitine to the auricle A purse-string suture was introduced into the auricular wall; an electrode was inserted through a small incision within the suture line and the sutures were drawn tight The electrode was placed on the septum close to the focus (Figure 119) and an electrocardiographic record was taken The electrode was then moved to a site on the septum as distal to the focus as possible and a second tracing was obtained (Figure 119). Here the intrinsic deflections in the direct leads fall in the trough of the flutter wave in a simultaneously recorded lead AVF in the same relative positions as observed in the tracings from the body of the auricle.

Observations similar to those described in experiment 1A have been made repeatedly during episodes of auricular paroxysmal tachycardia. Here again, the P' wave may be approximately outlined by simultaneous recording of two direct auricular leads and an indirect limb lead (Figure 120).

The intrinsic deflections inscribed from the electrode placed close to the aconitine focus must signify or immediately follow the onset of auricular depolarization. The intrinsic deflection inscribed from the electrode distal to the focus must coincide with or immediately precede the termination of auricular depolarization. The two deflections roughly mark the beginning and end of the auricular excitation wave; the time consumed as the impulse travels from the first to the second electrode represents almost the full duration of auricular excitation.

Experiment 1B: Demonstration of the Excitation Wave in Flutter by Means of Semi-Direct Leads in Man

Information derived from Experiment 1A may be utilized to outline the excitation wave in limb leads from human subjects in which this portion of the flutter complex is not clearly defined. Ideally, direct auricular leads should be employed as in the animal experiments. From a practical standpoint, the same results can be obtained with esophageal leads (Figure 121) and occasionally with precordial leads from areas directly overlying the auricles.

By means of auricular esophageal leads or auricular precordial leads taken simultaneously with indirect limb leads, observations were made in 10 patients during episodes of flutter. In each instance auricular intrinsic deflections could be clearly identified in the esophageal leads It was thus demonstrated that in man, as in the dog, the auricular intrinsicoid deflection occurs during the interval corresponding to

CHAPTER VII

An Electrocardiographic Analysis of the Structure of the Auricular Deflections of Auricular Flutter and Tachycardia

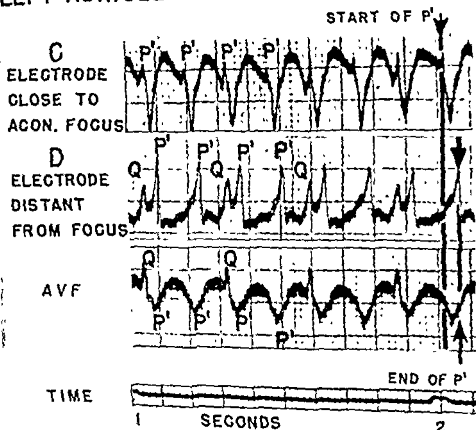
INTENSIVE study has shown that the ventricular complex consists essentially of waves of depolarization and repolarization. The auricular complex, unlike the ventricular deflection, has received little attention from electrocardiographers and the nature of its component parts is not as well understood. This is especially true in tachycardia and flutter, where the basic phenomena of auricular depolarization and repolarization have been completely ignored.

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The familiar continuous undulations of the flutter waves apparently have induced clinicians and electrocardiographers to accept the circus movement theory of flutter. This is understandable for such undulations might well be interpreted as a graphic representation of waves traveling in a circular pathway. If the flutter waves did not exhibit an undulating appearance, it is doubtful that the circus movement theory would have achieved general acceptance, especially in view of the admittedly incomplete supporting evidence.

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The electrocardiographic evidence presented in this chapter will show that the general principle of depolarization and repolarization applies also to flutter and tachycardia. The "F" wave of flutter is not a unique wave unrelated to other electrocardiographic deflections, but is probably composed of two components, namely, a wave of depolarization followed by a wave of repolarization. This undulating type of auricular complex representing the same phenomena may also occur in auricular paroxysmal tachycardia. The wave of depolarization will be considered in Part I of the chapter and the wave of repolarization in Part II.



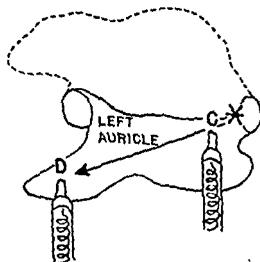


Figure 118. An experiment similar to that in Figure 117 except that … the left auricle Electrode C is close … focus Note that the intrinsic deflec…

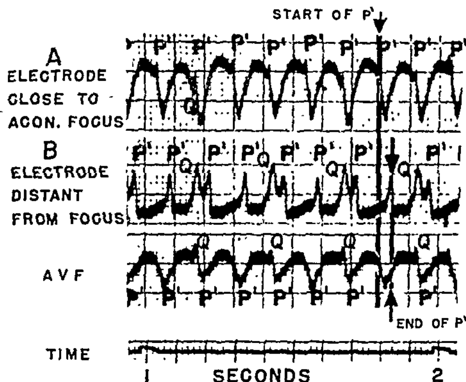


Figure 117. *Spread of flutter wave in right auricle of dog Aconitine* focus at caudal end Electrode A close to the focus, electrode B distant from the focus, both records were taken simultaneously with lead AVF The intrinsic deflection in A (arrow) denotes the start of the P' wave, the intrinsic deflection in B (arrow) denotes the end of the P' wave in lead AVF. Thus, *with the focus at the caudal end of the auricle, the P' wave in* lead AVF is the portion of the flutter complex delineated by the sharp downward deflection

the P' wave of the flutter complex in the indirect limb lead (Figure 122).

Experiment 2. Similar Nature of the Excitation Waves in Tachycardia an Flutter

This experiment demonstrates the progressive change in the configuration of the auricular deflections in limb leads which occurs with an increase in the rate of auricular contraction.

Auricular tachycardia was produced by the local application of aconitine to the surface of the body of the right auricle adjacent to the entrance of the inferior vena cava. Continuous *simultaneous limb lead electrocardiograms* were taken As the rate of discharge from the aconitine focus spontaneously increased, the typical flutter wave pattern gradually evolved (Figure 123). Usually the transition from the tachy-

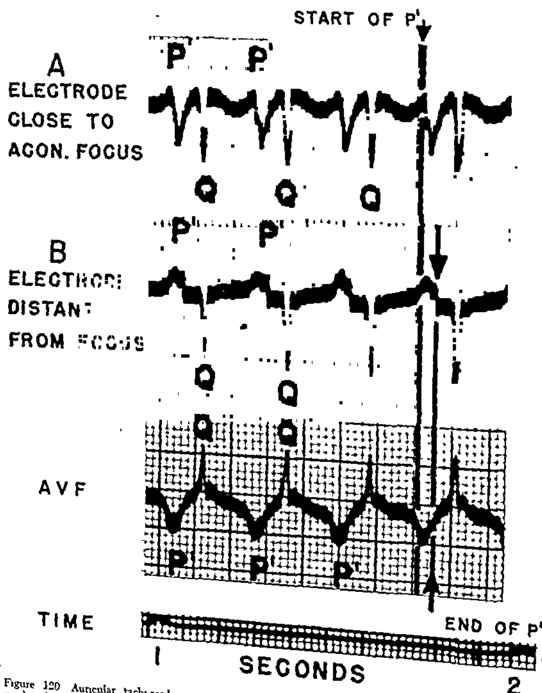


Figure 120 Auricular tachycardia experimentally produced with aconitine placed at the caudal end of the auricle (A) Electrode close to aconitine focus; (B) electrode distal to focus, simultaneous lead AVF. The intrinsic deflections from the direct auricular leads delineate the P' waves in lead AVF.

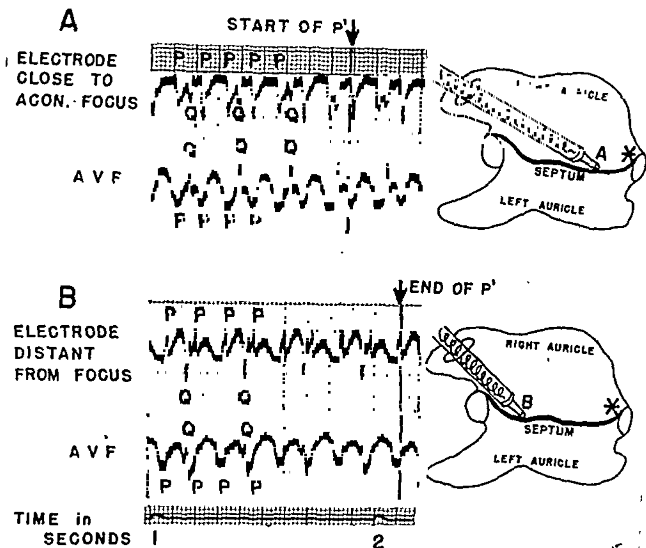


Figure 119. Transmission of the flutter wave along the interauricular septum. Aconitine focus placed at caudal end of auricle. An electrode was placed through a purse-string suture on the auricular wall and its tip placed against the septum. Simultaneous lead AVF was taken In A, the electrode is near the focus at the caudal end of the septum and the intrinsic deflection recorded denotes the beginning of the P′ wave. In B, the electrode has been moved to the cephalic portion of the septum The intrinsic deflection inscribed here denotes the end of the P′ wave Thus it is shown that the spread of the impulse in the septum is the same as in the bodies of the auricles and that the intrinsic deflection from each septal lead falls in the trough of the flutter wave in lead AVF in the same relative positions as in the previous experiments (Figures 117 and 118).

cardia wave to the flutter wave was gradual; occasionally it was abrupt. During the transition a complete series of configurations ranging from the sharp deflections of the tachycardia waves to the typical saw-tooth undulations of flutter was observed.

As seen in Figure 123A, at the onset of paroxysmal tachycardia the auricular rate is 187 beats per minute; the P′ wave is sharply inverted in lead 3, and is followed by a distinct isoelectric period. As the rate of auricular contraction increases to 320 beats per minute (Figure 123B) the inverted P′ wave is followed by an upright Ta wave. As the rate increases further, 2:1 auriculo-ventricular block develops, and the electrocardiogram becomes completely characteristic of flutter (Figure 123C). As the tachycardia rate increases, the P′-R interval becomes greatly prolonged (Figure 123). The sharply inverted auricular deflections seen initially in the episode of auricular tachycardia are clearly discernible in the subsequently developed flutter complexes; this sharply inverted portion of the flutter wave obviously is the excitation wave

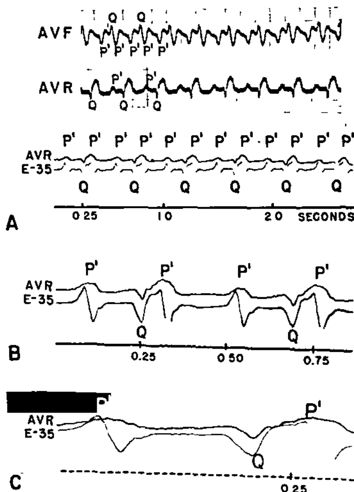


Figure 122 The upper tracings are leads AVF and AVR (not simul-taneous) from ... direct-writing ... esophageal lea... cathode-ray os... (B) faster speed, and (C) fastest speed used on this subject. Note that the intrinsicoid deflection in the esophageal lead clearly falls within the P' wave of AVR (oscillograph records slightly reduced).

bout of tachycardia The occurrence of both arrhythmias in the same patient is unusual, although similar examples have been described by Parkinson and Mathias,[490] Carr,[88] Barker and co-workers,[24, 25] Decherd and Herrmann,[133] and others.

The depolarization wave of clinical flutter can be demonstrated easily and convincingly in those instances in which one of the limb leads exhibits a depolarization wave without a Ta wave, this occurs most frequently in lead 1 (Figure 128). When such a lead 1 is obtained with simultaneously recorded leads 2 and 3 showing typical flutter undulations, the excitation wave in leads 2 and 3 may be identified by projection of the same wave from lead 1. Unfortunately, only rarely do clinical electrocardiograms of flutter lend themselves to such analysis

Experiment 2 and its clinical counterpart demonstrate that the typical undulatory wave of clinical flutter is not a unitary deflection specific for this arrhythmia and representative of constant auricular circus activity, but actually includes in its composition a segment identical with the P' wave of tachycardia. On the basis

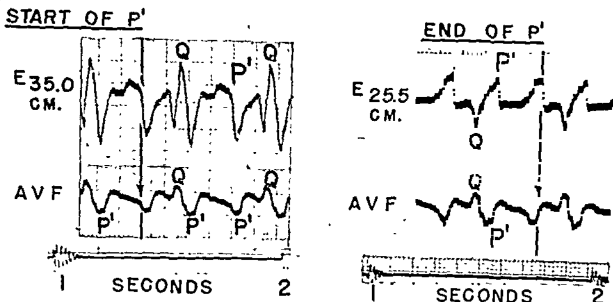


Figure 121 Esophageal lead and lead AVF taken simultaneously from a patient with auricular flutter (auricular rate 300 beats per minute). E-35 is an esophageal lead recorded 35 cm from the lips, at or near the level of the ectopic focus The downward deflection (arrow) is the intrinsicoid deflection and marks the onset of P' wave in lead AVF. Right Esophageal lead recorded 25 cm from lips, corresponding to the cephalic end of the auricles, the intrinsicoid deflection (arrow) is inscribed approximately 0 04 second later than in E-35 and marks the end of the P' wave in lead AVF, thus delineating the P' portion of the flutter complex in the human

of flutter. When both disorders arise from the same ectopic focus, *the P' wave of tachycardia and the excitation wave of the flutter complex are similar in contour.*

When the aconitine focus is placed at or near the sino-auricular node, the appearance of the P' wave does not clearly show the time of onset of the arrhythmia since there is little or no change in the configuration of the auricular deflections. In such experiments the transition from sinus rhythm to auricular paroxysmal tachycardia is indicated by a sudden, though slight, change in auricular rate (Figure 124). As the tachycardia becomes progressively more rapid, an auricular rate is reached (in this experiment 320 beats per minute. Figure 124) at which flutter with 2:1 block develops The depolarization waves of both arrhythmias are again similar in contour and closely resemble those of normal sinus rhythm.

A flutter rhythm often can be reverted to auricular paroxysmal tachycardia by freezing the ectopic focus; when the focus is allowed to return toward body temperature flutter may reappear. Under such circumstances electrocardiograms demonstrate all possible transitional forms between the sharp configuration of the P' wave of tachycardia and the saw-tooth undulations of flutter (Figure 125).

During the transition from tachycardia to flutter, progressive auriculo-ventricular block occasionally appears. The change is analogous to the Wenckebach phenomenon; the P'-R interval gradually lengthens until dropped beats occur (Figure 126). In these instances, the progressive block occurs while the auricles are contracting at a relatively rapid rate. The reverse changes have been seen in transitions from flutter to tachycardia

The shape of the excitation waves during the transition from tachycardia to flutter remains the same.

Clinical Counterpart: Figure 127 illustrates electrocardiograms from a patient with bouts of both auricular paroxysmal tachycardia and auricular flutter which arose at different times, apparently from the same ectopic focus. *The excitation waves of both disorders are similar in shape* This appears to be an exact clinical counterpart of experiment 2 The depolarization wave in flutter can be recognized with the same degree of certainty as the P' deflection in the

Figure 124 Records from simultaneous leads 1, 2, and 3 at double EKG speeds. Aconitine was placed near the sinus node in a dog In A, the auricular paroxysmal tachycardia starts at the 4th beat as signified by a slight but definite increase in rate from 118 to 122 beats per minute and a slight change in the shape of the auricular deflection In B, the rate gradually increases to 200 beats per minute. In C, 2·1 auriculo-ventricular block occurs at an auricular rate of 320 beats per minute *Note that after the onset of tachycardia the shape of the P' wave remains constant throughout the record*

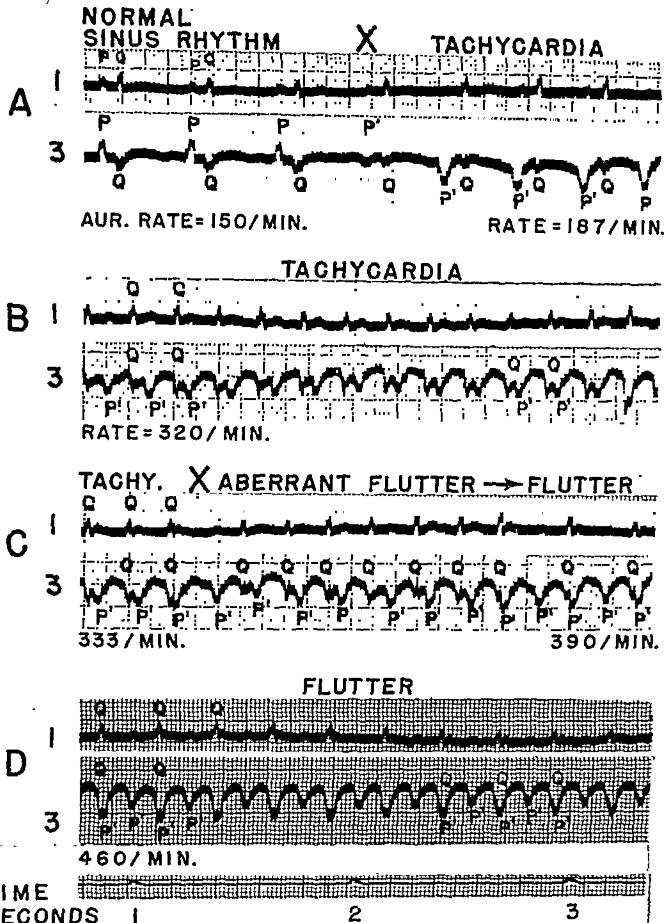


Figure 123 Simultaneous leads 1 and 3 Aconitine placed at caudal end of auricle in a dog In A, auricular paroxysmal tachycardia starts at a rate of 187 beats per minute (X). With the onset of tachycardia, the upright P wave of *sinus rhythm* has become inverted (P' wave) in lead 3 In B, the tachycardia increases in speed to 320 beats per minute. In C, the flutter appears at X and rapidly becomes 2 1 *flutter at an auricular rate of* 390 *beats* per minute at the end of the strip In D, 2 1 flutter continues as the rate increases to 460 beats per minute *After the onset of tachycardia the appearance of the P' wave remains the same throughout the record.*

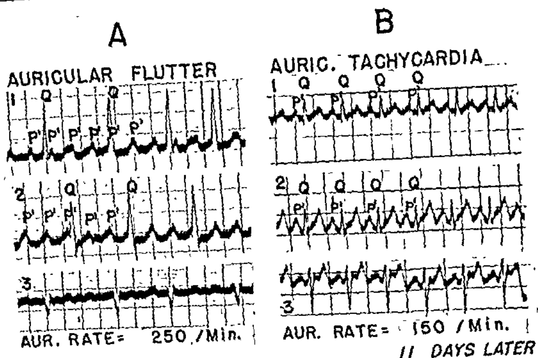


Figure 127. (A) Electrocardiogram of auricular flutter in man with upright P′ waves in leads 1 and 2. Auricular rate is 250 beats per minute.
(B) Eleven days later, the patient had auricular tachycardia at a rate of 150 beats per minute.
The P′ waves are identical in shape in both instances.
The occurrence of these two arrhythmias in the same patient is comparatively rare.

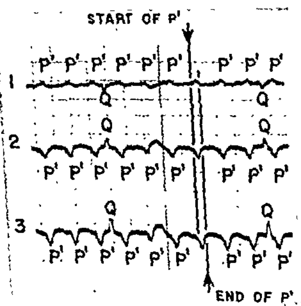


Figure 128. Simultaneous electrocardiograms from leads 1, 2 and 3. In lead 1 a pure wave of depolarization (P′) is seen. No Ta wave is apparent. When the beginning and end of the P′ wave is superimposed on leads 2 and 3 which show a continuous undulation, the wave of depolarization (P′) can be easily outlined.

Experiment 4: The Effect of Change in the Site of the Ectopic Focus Upon the Shape of the P′ Wave in Flutter

The shape of the P′ wave in auricular premature systole and in auricular paroxysmal tachycardia varies with the site of the ectopic focus in the auricles; this is true of both man and animals (Chapters II, III and IV). Furthermore, the contour of the P′ waves arising from a given ectopic focus is similar whether the arrhythmia is auricular tachycardia or flutter (experiment 2). Thus it would follow that the configuration of the P′ waves of flutter also varies with the location of the experimentally produced ectopic focus. Because of the observed changes in the auricular complexes caused by differences in the position of the heart in the thorax, these observations obviously must be made on the same animal. This was successfully done in the following manner on four occasions.

Aconitine was placed on the tip of the right

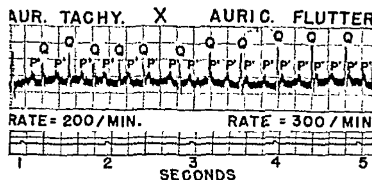


Figure 125. Increase in auricular rate following warming of the aconitine focus to body temperature after it had been frozen in a dog At the start of the record the auricular rate is 200 beats per minute and the tracing is typical of flutter The P' wave is identical throughout. These rapid changes in rate, produced artificially in these experiments by changing the temperature of the ectopic focus are instructive, however, clinically, such rapid changes in rate do not occur spontaneously.

of this observation, together with the results of Experiment 1, *it is concluded that the segment in question represents the period of depolarization of the auricle and therefore may properly be called the P' wave of flutter.*

Experiment 3: Effect of Positional Change on the Auricular Deflections of Flutter

This experiment illustrates that the typical undulatory pattern of the auricular electrocardiogram is not an essential characteristic of flutter.

During a single bout of experimental flutter the position of the heart in the thorax was changed manually several times in various directions. Each positional change was reflected in variations of the "flutter" configuration in the limb lead electrocardiogram (Figure 129). The experiment was repeated in five animals.

Clinical Counterpart: Similar observations have been made on three patients with auricular flutter. Electrocardiograms were taken continuously while the position of the patient was varied. Figure 130 shows lead AVL from one patient. When the patient is lying on his right side the flutter wave is indiscernible. When the patient is supine the flutter waves are difficult to recognize. When the patient is lying on his left side or is sitting the flutter waves are readily seen.

We have observed several instances in which the typical flutter waves disappeared or became greatly modified with deep inspiration (Figure 131). The change in the position of the auricles in relation to the limbs, resulting from the lowering of the diaphragm, probably accounts for these electrocardiographic differences.

It is thus shown that the saw-tooth undula*tions long considered essential to the electro*cardiographic diagnosis of flutter can be made to appear and disappear at will by varying the position of the heart in the thorax. Hence, *the presence of these undulations cannot be considered an essential criterion for the diagnosis of flutter.*

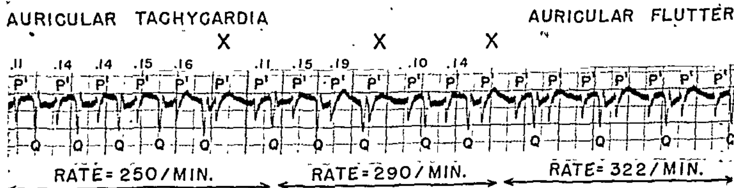


Figure 126 From a direct auricular lead in a dog The auricular rate becomes more rapid as the previously cooled aconitine focus is warmed to body temperature The first fourteen beats are typical of auricular paroxysmal tachycardia with progressive auriculo-ventricular block and dropped ventricular beats at X (Wenckebach phenomenon) As the rate increases from 250 to 322 beats per minute 2 1 flutter appears The P' wave is identical throughout P'-R intervals are inscribed above the tracing Tracing recorded at 50 mm per second

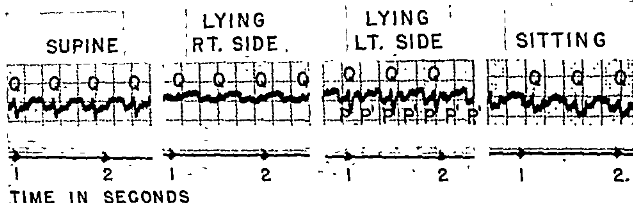


Figure 130 Lead AVL from a patient with 2 1 flutter, effect of position of heart on the flutter deflections The flutter wave can be clearly seen only with patient lying on left side, the auricular deflections are indistinct or invisible in other positions

dix, the P′ wave throughout the record, in both the episodes of tachycardia and those of flutter, is a sharp upright deflection in leads 1, 2 and 3 closely resembling the P wave of normal sinus rhythm. In the electrocardiograms taken with the ectopic focus at the caudal end of the same auricle, the P′ wave is sharply inverted in leads 2 and 3 in both tachycardia and flutter.

Thus, the shape of the P′ waves of auricular tachycardia and those of auricular flutter vary in a similar manner with the change in location of the initiating ectopic focus. In the present experiments, when the ectopic focus is at the tip of the right auricular appendix, the P′ wave is upright, when the initiating focus is at the caudal extremity of the auricle, the P′ wave is sharply inverted. Furthermore, when both arrhythmias arise from the same focus in the same auricle, the P′ waves of tachycardia and flutter are of similar configuration.

Clinical Applications: The observations that the P′ wave can be identified in tachycardia and in flutter and that its shape varies with the site of the ectopic focus in the auricles have proved to be of clinical significance. In any *given instance* of tachycardia or flutter, a certain configuration of the P′ wave implicates a particular site in the auricle as the location of the *initiating* focus, as shown in auricular premature systole (Chapter IV), any change in that configuration is due to a shift in the location of the focus.

Summary: Evidence has been presented demonstrating that (1) the first part of the flutter wave actually consists of a P′ wave, the wave of excitation or depolarization; and (2) the shape of the flutter wave varies with its site of origin and with the position of the heart in the chest. These conclusions apply to both man and animals.

Part II

DEMONSTRATION OF THE Ta WAVE IN FLUTTER AND TACHYCARDIA

In Part I of this chapter, it is clearly demonstrated electrocardiographically that a P′ wave is a component of the auricular complex in both experimental and clinical flutter. Evidence will be presented to *show that the P′ wave in flutter* is generally followed by a large, easily visible, usually oppositely directed Ta segment or wave (sometimes called the P-Ta segment). As noted earlier, in normal sinus rhythm the P wave may be followed by a Ta wave which represents re-

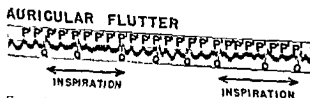


Figure 131 The effect of deep inspiration in a patient with flutter. The flutter undulations become indistinct during inspiration (due to the descent of the diaphragm and consequent change in position of the heart)

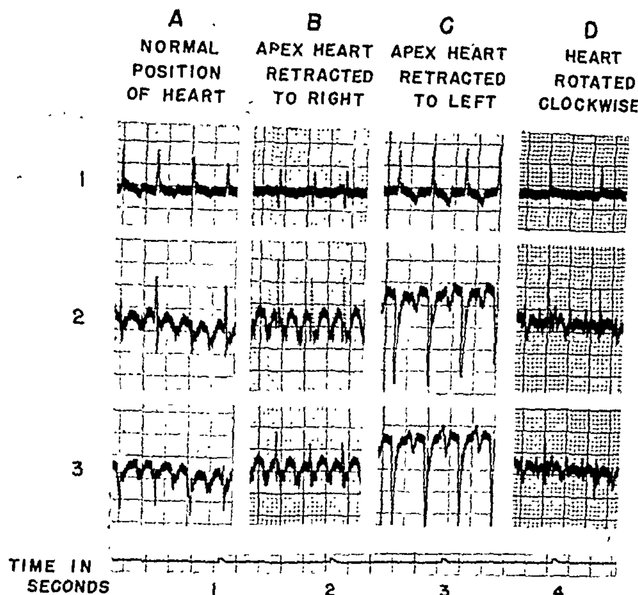


Figure 129. The effect of changing the position of the heart on the auricular deflections of flutter in a dog.

(A) Normal position of the heart Typical 2 1 flutter is present with undulating waves

B, C and D are records obtained after the heart was rotated manually in various positions When the heart is retracted to the left (C), and rotated clockwise (D), the flutter undulations become indistinct and finally disappear. This clearly shows that the flutter configuration can be made to disappear merely by changing the position of the heart

auricular appendix and auricular paroxysmal tachycardia was produced (Figure 132A). Continuous simultaneous electrocardiograms were made of limb leads 1, 2 and 3. As the rate of discharge from the focus spontaneously increased, the electrocardiogram became characteristic of flutter (Figure 132B).

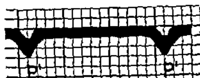
The aconitine focus at the tip of the right auricular appendix was then frozen with ethyl chloride spray and was maintained in a frozen state for the remainder of the experiment. This procedure prevented stimulus formation in the appendical focus and abolished its pacemaking function; sinus rhythm supervened.

Aconitine was then placed at the caudal end of the right auricle in the same animal, just ventral to the entrance of the inferior vena cava (Figure 132C); again, auricular paroxysmal tachycardia appeared. As the auricular rate gradually increased, 2:1 auriculo-ventricular block developed and the electrocardiogram became characteristic of flutter (Figure 132D).

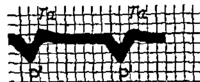
The electrocardiograms show that when the ectopic focus lies on the right auricular appen-

AURICULAR RATE / MIN.

100



150



188



300



375



Figure 138 Semi-diagrammatic representation of the evolution of the Ta wave in auricular flutter At an auricular rate of 100 beats per minute there is an absolutely flat isoelectric line between successive P' waves and no Ta deflection is seen At 150 beats per minute a very small but perceptible Ta wave appears. At a rate of 188 beats per minute, the Ta wave is distinct but is followed by an isoelectric interval At a rate of 300 beats per minute the Ta wave is larger than before and only a short isoelectric "shelf" follows the Ta wave At a rate of 375 per *minute*, the Ta wave is large and no isoelectric period is present. This record shows a characteristic sine wave *typical of* flutter produced at the caudal end of the auricle as recorded from leads 2 and 3 All of the configurations schematically shown in this illustration have been seen in *electrocardiograms* in both man and animals

occurrence following the P' wave of auricular excitation strongly suggests that it is concerned with auricular repolarization. As the auricular rate reaches 375 beats per minute, the Ta wave becomes still larger and finally attains an amplitude of 0.3 millivolts; 2:1 auriculo-ventricular block appears and the electrocardiogram is typical of flutter (Figure 134D). It is the Ta wave which gives flutter its undulating appearance, for little significant change occurs in the P'wave during the transition from tachycardia to flutter. The Ta wave becomes so large and bowed that it is often the most prominent part of the flutter complex and is more distinct than the P' wave.

The Ta wave of repolarization apparently is induced or rendered visible by the more rapid rate of auricular contraction and progressive prolongation of the P'-R interval. The faster the auricular rate, the larger is the Ta segment. The Ta wave causes some distortion of the succeeding QRS complex.

Clinical Counterpart (Effect of Rate on the Ta Wave): Examination of a large number of electrocardiograms of patients with flutter reveals that when the auricular rate is slow, the P' wave is often followed by an isoelectric period; with more rapid auricular rates, the P' waves are usually followed by large, oppositely directed Ta deflections (Figures 135 and 136). This relationship is even more strikingly illus-

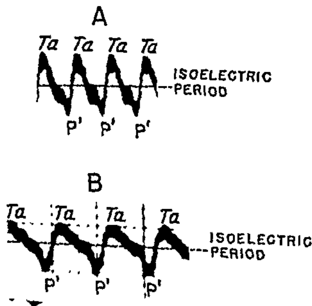


Figure 139 Enlargement (three times) of records of flutter complexes to show that the Ta wave is in a direction opposite to that of the P' *wave* The Ta wave starts immediately after and as a continuation of the upstroke of the P' wave A short *isoelectric* period follows the Ta wave. The auricular rate is 400 beats per minute in A and 280 beats per minute in B.

Figure 135. Records from the same leads in two patients with flutter.

(A) rate 200 per minute. No Ta waves are present.

(B) rate 330 beats per minute. The P′ waves are of the same shape as in A; at this rapid rate large Ta waves have appeared.

Observation 1: Evolution of the Ta Wave as the Rate of Auricular Contraction Increases

The evolution of the Ta wave as the auricular rate increases during the transition from tachycardia to flutter may be seen in Figure 123 as well as in the following experiment.

Aconitine was placed on the caudal end of the right auricle and the resulting transitions from normal sinus rhythm to tachycardia to flutter recorded (Figure 134). The P wave of normal sinus rhythm is upright in lead three (Figure 134A). As auricular paroxysmal tachycardia occurs at a rate of 220 beats per minute, deeply inverted P′ waves appear and are followed by isoelectric periods or slight upward undulations. As the rate of auricular contraction increases to 264 per minute, the isoelectric period is replaced by an upward bowing immediately following the inverted P′ deflection (Figure 134C). Since the deflection starts immediately after the P′ wave and precedes the ventricular QRS deflection, it obviously represents auricular and not ventricular activity, its

Figure 136 (A) Record from a patient with auricular flutter. Rate 200 beats per minute

(B) Record from another patient with auricular flutter Rate 300 beats per minute

Distinct undulations due to large Ta waves are present in B. In A, the Ta waves are absent or very small Thus, in man, as in the dog, development of the Ta wave depends largely on the auricular rate

Figure 137. Flutter at different rates in a single patient

(A) Auricular rate 250 beats per minute Note isoelectric intervals or only very small Ta waves after each P′ wave.

(B) Same patient, seven days later Rate 300 beats per minute Ta waves have appeared and are responsible for the typical undulatory, flutter appearance.

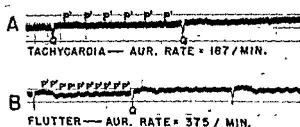


Figure 142 Records from a patient with supraventricular tachycardia and complete auriculo-ventricular block, lead 2.

(A) Auricular rate 187 beats per minute, a small upright P′ wave is present

(B) The auricular rate has increased to 375 beats per minute The P′ wave is still small and upright

Undulations are present in B but not in A Thus, A might be called auricular tachycardia, and B might be called auricular flutter, however, in view of the facts concerning the development of the Ta wave as set forth in this study, this distinction would appear to be without significance

are illustrated diagrammatically in Figure 138. When the auricular rate is relatively slow, only the inverted P′ wave is seen, no Ta wave is discernible. As the rate increases, the upstroke of the P′ wave continues above the isoelectric level to become the upstroke of the Ta wave; the downstroke of the Ta wave then descends in a relatively gentler slope back to the isoelectric line The upstroke of the Ta wave is thus a direct continuation of the upstroke of the P′ wave. As the auricular rate further increases, the Ta wave becomes larger and more bowed, and the isoelectric period becomes shorter, *this isoelectric period may sometimes be seen as a short shelf between the end of the Ta wave and the*

...bowed, the isoelectric "shelf" completely disappears, and the more gradually descending limb of the Ta wave directly joins the descending limb of the succeeding P′ wave

When the flutter rate is unusually slow or when the flutter impulse starts near the sinus node, the Ta wave may be small or entirely absent Distinct isoelectric periods often are present and such tracings frequently are inter-

...ation of the form and evolution of the Ta wave as set forth in the preceding paragraphs shows the meaninglessness of such a distinction.

Examination of a large number of records of experimental and clinical flutter and tachycardia reveals that in limb leads the Ta wave is usually inscribed immediately after and in a direction opposite that of the P′ wave; * no isoelectric period occurs between these two deflections (Figure 139).

Observation 3· Rapid-Rate Auricular Tachycardia with Ta Waves

Attention has previously been called to the fact that rapid auricular tachycardias (occurring in infants and occasionally in adults) may exhibit deflections in which the two component parts, P′ wave and large Ta wave, can probably be clearly identified (Figure 140). Because of their undulatory appearance, such tracings are often diagnosed as auricular flutter with 1:1 auriculo-ventricular response. Again, in view of the foregoing discussion of the evolution of the Ta wave, this distinction is without significance.

Unfortunately, in most cases of auricular tachycardia the auricular events are completely masked by the ventricular complexes, especially by the large ventricular T waves. In these instances an exact diagnosis is impossible. In many electrocardiograms of rapid rate tachycardia, the P′ waves cannot be identified with certainty without the aid of direct leads or special semi-direct leads, such as esophageal or chest leads.

Figure 141 shows electrocardiograms of tachycardia and flutter produced experimentally in the same animal. No auricular activity is evident in the limb leads, however, simultaneous direct auricular leads clearly show P′ waves. Lead AVF is almost identical in tachycardia and flutter.

Observation 4: The Auricular Deflection in Clinical Tachycardia and Flutter with Complete Auriculo-Ventricular Block

During the progress of the investigation seven patients were encountered whose electro-

* One example has been found in which the P′ and Ta waves are in the same direction (Chapter X).

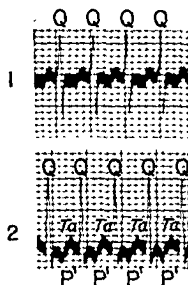


Figure 140. Records from a child, age two, with auricular paroxysmal tachycardia at rate of 150 beats per minute, enlarged two times. Deeply inverted P′ waves can be seen in lead 2 and appear to be followed by upright Ta waves

trated *in electrocardiograms of flutter at different* rates in the same patient (Figure 137). When the auricular rate is 250 beats per minute (Figure 137A), the P′ wave alone is present and is followed by a distinct isoelectric period. Seven days later, for unknown reasons the auricular rate had increased to 300 beats per minute (Figure 137B); definite Ta waves seen best in leads 2 and 3 are now present and the record has the undulating appearance characteristic of flutter. In the *electrocardiograms recorded on* both dates the P′ deflections are identical, indicating that the same ectopic focus initiated the arrhythmia in each instance.

In both man and animals, when the auricular rate is relatively slow, Ta waves are absent; when the rate is rapid, Ta waves are likely to be present. It is therefore apparent that *in the* flutter wave of an uninjured auricle, free from effects of drugs, the presence or absence of a characteristic Ta wave is largely dependent on *the auricular rate.*

Observation 2: Direction and Form of Ta Waves in Flutter and Tachycardia

The increase in size and change in form of the Ta wave which occur with the progressive increase in auricular rate (Figures 136 and 137)

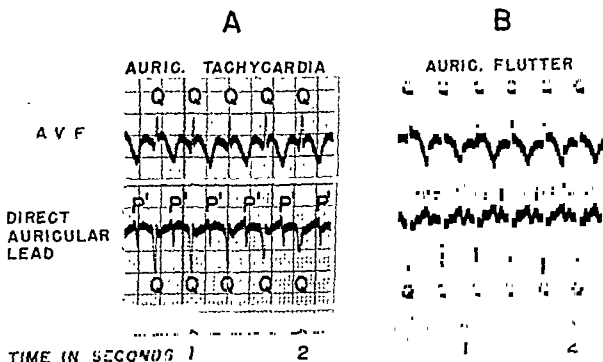


Figure 141. Electrocardiograms of aconitine induced auricular tachycardia (A) and flutter (B) in dogs. Note absence of auricular activity in lead AVF, while simultaneous direct auricular lead clearly reveals auricular deflections. Direct leads in A and B recorded from different sites on auricle

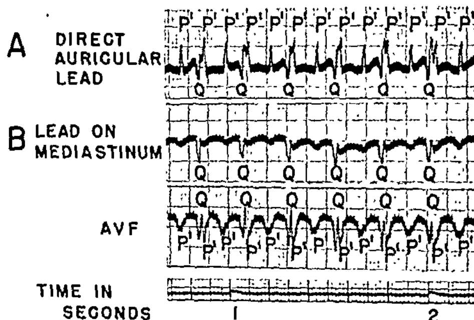


Figure 145. Variations in the configuration of the flutter deflections due to differences in the location of the electrode with reference to the auricle.

(A) Direct auricular lead (in dog). Note distinct P′ waves with isoelectric intervals

(B) Lead from mediastinal structures at some distance from the auricle taken simultaneously with direct auricular lead

In AVF taken simultaneously

Thus the presence or absence … location

waves. The similarity of this electrocardiogram, identified as flutter, to some of the leads in Figure 142, called tachycardia, *is striking*, the only difference is that of rate.

In summary, a study of tracings from patients with heart block accompanied by regular auricular arrhythmias demonstrates that no sharp line of demarcation exists between the electrocardiographic configurations of flutter and tachycardia.

Observation 5 Esophageal and Precordial Leads

Brown[74] reported differences between the auricular deflections of auricular paroxysmal tachycardia and those of flutter in esophageal leads In four instances of auricular flutter he observed undulating, never-resting waves with sharp peaks On the other *hand*, in tracings from two patients with auricular paroxysmal tachycardia, relatively long isoelectric intervals *were present.* Brown suggested that a dissimilarity in the fundamental mechanism of the two arrhythmias might account for these electrocardiographic differences.

In order to properly interpret esophageal electrocardiograms in auricular paroxysmal *tachycardia* and flutter, the following considerations must be understood. In dogs with flutter, *the deflections in direct* auricular leads differ from those in esophageal leads *in that the* former exhibit definite isoelectric intervals between the auricular complexes (Figure 145). *Semi-direct* leads, such as those taken *from the* mediastinal structures (comparable to esopha*geal leads*), may exhibit intermediate types of deflections; for example, spikes *similar to* those from direct leads may be present but with undulations instead of *isoelectric* periods (Figure 145). *Indirect* limb leads *show* the familiar flutter undulations without evidence of intrinsic deflection.

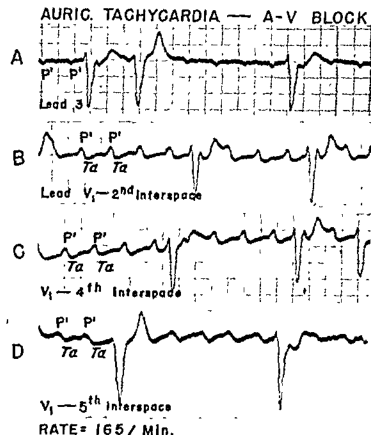


Figure 143. Electrocardiogram from a patient during a single bout of an auricular arrhythmia at a rate of 165 beats per minute with complete auriculo-ventricular block following a posterior coronary occlusion.

In lead 3 (A) inverted P' waves are present, there is no Ta wave In lead V_1 in the 2nd interspace (B), in lead V_1 in the 4th interspace (C), and in lead V_1 in the 5th interspace (D) large Ta waves are present and impart to the tracing an undulatory, flutter-like appearance despite the comparatively slow rate Thus, in a single record the Ta wave may be present in some leads and absent in others, when present it gives the tracing the typical undulating appearance of flutter

Some would diagnose the same arrhythmia as recorded in A as tachycardia and as recorded in B, C and D as flutter

cardiograms exhibited complete auriculo-ventricular dissociation (with ventricular rates under 50 beats per minute) and regular auricular arrhythmias of various types. An opportunity was thus afforded for observation of pure auricular deflections from ectopic foci unaltered by ventricular complexes Examination of these tracings confirms the fact that no sharp demarcation exists between the electrocardiographic configuration of flutter and that of tachycardia.

In one patient with complete auriculo-ventricular block, regular auricular rhythms of varying rates arose from the same ectopic focus at different times (Figure 142). In Figure 142A, the rate is 187 beats per minute and distinct isoelectric periods are present; such a record has been called auricular tachycardia. In Figure 142B the auricular rate has spontaneously increased to 375 beats per minute but now distinct undulations are present; because of the more rapid rate and the presence of the undulations this record would be termed flutter. Actually these two records represent the same auricular disturbance arising from the same ectopic focus with different auricular rates.

Figure 143 is a record from a second patient with complete auriculo-ventricular block and regular auricular tachycardia; the auricular rate is 165 beats per minute. In A (lead 3) inverted P' waves are present and Ta waves are absent. In D (*lead* V_1*, 5th interspace) large* Ta *waves are* present, imparting the typical undulatory appearance of flutter. In B (V_1 2nd interspace) and C (V_1, 4th interspace) intermediate types of deflections are recorded. Thus in a given episode of an arrhythmia at a constant rate, Ta waves may be present in some leads and absent *in others. When the Ta waves are present, even* though the rate is relatively slow, the configura*tion is identical with that of flutter*

Figure 144 is a tracing from a patient with complete auriculo-ventricular block and auricular flutter with a rate of 214 beats per minute. The flutter undulations are present in lead 2; *lead AVL shows only pure P' waves without Ta*

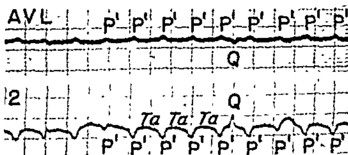


Figure 144 Tracings from a patient with complete auriculo-ventricular block and auricular flutter with an auricular rate of 214 beats per minute The flutter undulations are present in lead 2, lead AVL shows only pure P' waves without Ta waves The record in lead 2 is characteristic of auricular flutter, the simultaneously recorded lead AVL might be called auricular tachycardia

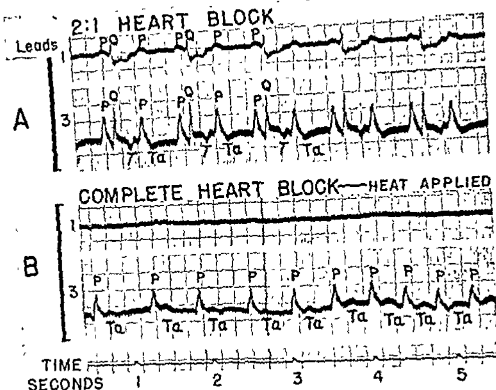


Figure 147. Ta wave due to auricular injury Leads 1 and 3. Heart block in a dog was established by cutting the Bundle of His

(A) With 2 1 heart block Ta waves are small, inverted, and inscribed in a direction opposite to that of the P' waves

(B) After complete heart block was established, hot lamp was placed a few inches away from auricles. Upright Ta waves appeared and became very marked The Ta waves after injury with heat (B) are in the same direction as the P' wave. In flutter the P' and Ta waves are oppositely directed.

tachycardia, also inscribed in a direction opposite that of the P' wave, may likewise represent the result of auricular "strain." *

The concept of auricular "strain" as a consequence of rapid auricular rates is confirmed by study of the cinematographs The films show that under such circumstances the auricles work hard and appear to be near the limit of co-ordinate contraction. The period of diastole is brief and catabolites in high concentration might be expected to collect as the result of insufficient rest Figure 150 shows the approximate duration of systole and diastole in flutter and various types of tachycardia. In flutter at the rate of 464 cycles per minute, systole occupies approximately 73 per cent of the cardiac cycle; in paroxysmal tachycardia at the rate of 198 beats per minute, the duration of systole is 41.4 per cent of the cycle; in sinus tachycardia at the rate of 164 cycles per minute, the duration of systole is 33 6 per cent. (These figures were obtained from high-speed cinematographs by measuring with a stopwatch the time occupied by systole from its onset to its maximum degree.)

If biochemical changes are associated with electrical events, the accumulation of catabolites may be responsible for the flutter Ta wave. Such abnormal conditions obviously would affect the process of repolarization of the auricular musculature. Ta waves may also occur in auricular infarction [121, 273, 269, 323, 327] and possi-

* The authors do not necessarily advocate the term "strain" for these types of electrocardiographic patterns; it is merely suggested that an analogy might exist between the Ta wave of auricular injury and the electrocardiographic pattern sometimes called "ventricular strain."

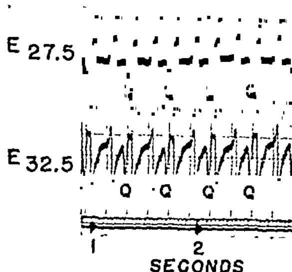


Figure 146 Esophageal leads recorded in a patient with 2 1 flutter. The record above is 27 5 centimeters from lips; that below is 32.5 centimeters from lips. E 27.5 shows upright P′ waves with isoelectric periods. In E 32 5 the undulations are present between the P′ waves. Thus the presence or absence of flutter undulations depends in part on the relation of the electrode to the auricles and may be present or absent in different esophageal leads in the same patient.

Esophageal leads cannot be regarded as direct leads; they are semi-direct leads, since the electrode is close to but not directly on the the auricles. Hence, flutter undulations would be expected to occur in esophageal leads on occasion but not as a regular rule; such has been the experience in this laboratory. In some instances in which the esophageal leads do not reveal undulations, simultaneous limb leads may exhibit typical flutter undulations. In flutter, esophageal leads at various auricular levels may show either typical flutter undulations or P′ waves with isoelectric intervals (Figure 146).

Thus, the presence or absence of an isoelectric period in esophageal leads does not provide a basis for the assumption that the mechanisms of tachycardia and flutter are different.

Observation 6· Ta Wave Due to Auricular Injury

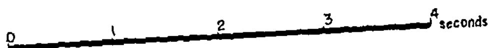
To facilitate the identification of the auricular deflections, auriculo-ventricular block was produced in a dog's heart by cutting the Bundle of His; normal sino-auricular rhythm was retained (Figure 147). The auricles were then injured with heat from a lamp. The effect of such injury is shown in Figure 147; the Ta wave becomes upright, progressively larger and more bowed. *In contrast to the Ta wave of flutter* which is usually in a direction opposite that of the P′ wave, the Ta wave after injury is generally in the same direction as the intrinsic deflection. This observation suggests that the mechanism of the Ta wave of flutter and tachycardia is different from that of the Ta wave caused by auricular injury with heat.

Another form of injury often observed experimentally is that produced by excessive pressure on the auricular wall by a direct auricular electrode. The resulting "current of injury" is illustrated in Figure 148. The appearance of large Ta waves in electrocardiograms of both dogs and man as a result of auricular injury has been described previously.[119]

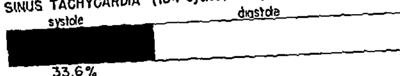
Clinical Applications: As previously mentioned, large Ta waves are occasionally seen during normal sinus rhythm in patients with severely damaged hearts. Figure 149 is a tracing from a patient with coronary arteriosclerotic heart disease recorded just before death. The P wave is followed by a large upright Ta deflection. The ascending limb of this deflection precedes the QRS complex; the descending limb causes considerable elevation of the ST segment. It is thus possible that ST segment *shift occasionally may be the result of distortion* by superimposed Ta waves; this has been suggested by other investigators. In most instances ST deviations due to auricular deflections can *be distinguished from those due to coronary* artery disease by the fact that in the former an additional auricular component is always present between the P wave and the QRS complex.

The foregoing discussion of the Ta wave suggests a possible analogy to the electrocardiographic pattern of "ventricular strain." In ventricular "strain" the T wave is almost always *inscribed in a direction opposite that of the* QRS complex. An auricle driven at a rate of 400 impulses per minute certainly is "strained"; the large TA deflection of flutter and rapid-rate

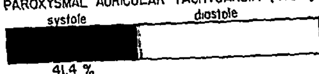
SYSTOLE — DIASTOLE RELATIONSHIP
in
TACHYCARDIA and FLUTTER



SINUS TACHYCARDIA (164 cycles/min·)



PAROXYSMAL AURICULAR TACHYCARDIA (198 cycles/min·)



FLUTTER (464 cycles/min·)

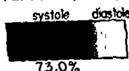


Figure 150 The relative ratios of systole and diastole in flutter Timing of each phase was done by stop watch while observing the high-speed cinematographs of the experimental ...

... minations made in different experiments They clearly demonstrate that as the auricular rate becomes faster the *ratio of systole to diastole* becomes greater The diastolic period of rest in flutter is very brief.

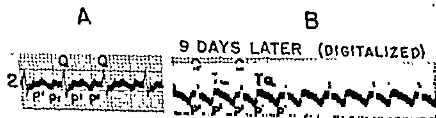


Figure 151 Effect of digitalis on flutter configuration

(*A*) Lead 2 from an undigitalized patient Note small Ta waves after inverted P' waves

(*B*) Nine days later, the same patient after digitalization Note that the Ta wave is much more distinct.

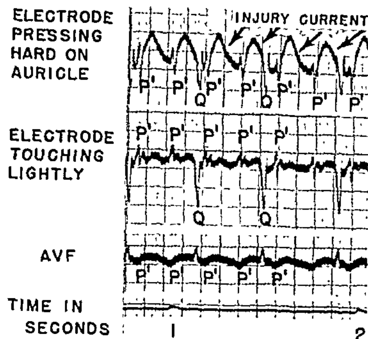


Figure 148. Ta waves of "current of injury" Two auricular leads and AVF were recorded simultaneously in a dog with flutter In the top tracing the electrode was pressed too firmly against the auricle and produced an "injury current." There is no "injury current" in the middle tracing where the electrode was gently pressed against the auricular surface Simultaneous AVF shows typical auricular flutter.

bly in azotemia. Although the mechanism of the production of the Ta wave in this latter condition is not definitely known, here also the mechanism may possibly be chemical injury from excessive accumulation of catabolites. This entire subject of the effect of auricular injury of various types [1] and of "strain" due to overwork,[656] on the formation of Ta waves requires further study.

Observation 7. Effect of Digitalis and Quinidine on the Flutter Deflection

In two patients with auricular flutter, the amplitude of the Ta wave increased following the administration of digitalis (Figure 151). These changes in the Ta wave of flutter are strikingly analogous to the effect of digitalis on the ventricular T wave. Digistalis produces deviation of the ventricular T wave in a direction opposite that of the QRS complex and shortens or completely abolishes the isoelectric period between these deflections. The increase in amplitude of the Ta wave has also been noted in the experimental animal (Figure 152).

The flutter wave is greatly widened and its rate slowed after quinidine administration (Figure 153). Other examples of the effect of quinidine on auricular flutter are seen in Figure 297, Chapter XVI. The effect of quinidine on the flutter wave as correlated with quinidine levels in the blood has been studied by Sokolow.[570]

SUMMARY AND CONCLUSION

The configurations of the auricular deflections of auricular paroxysmal tachycardia and flutter have been analyzed.

In man and in animals, in both arrhythmias the auricular deflection probably consists of two parts: (1) the P′ wave of depolarization and (2) the Ta wave of repolarization. The Ta wave becomes clearly visible as the auricular rate increases and, at rapid rates, imparts to the electrocardiogram the familiar undulations usually regarded as characteristic of flutter.

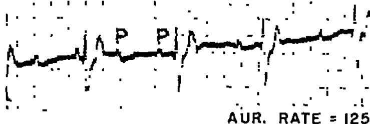
Identification and delineation of the P′ wave of the flutter complex in limb leads have been achieved by means of simultaneously recorded limb and direct auricular leads. In a given auricle, changes in the location of the ectopic focus result in alterations in the shape of the P′ wave. When the ectopic focus is in the region of the sinus node or the right appendix, the P′ wave in leads 2 and 3 is upright; when the focus is at the caudal end of the auricle, the P′ wave in leads 2 and 3 is inverted. Continuous tracings made during the transition from tachycardia to flutter show that the initial deflection (the P′ wave) maintains a similar appearance throughout the record. Thus, from a constant focus in a given animal, the shape of the P′ wave is essentially the same in the two arrhyth-



Figure 149 Lead 3 from a patient with arteriosclerotic heart disease and profound shock The electrocardiogram was taken shortly before death Note that the Ta wave starts just after the P wave, the elevated Ta wave causes some distortion of the QRS and the first part of the RST segment Ta waves are often found after auricular infarction

esophageal (semi-direct) leads in man has been considered. During the rapid auricular arrhythmias the P' and Ta waves may be present in esophageal leads, however, isoelectric periods are not uncommon in these leads.

Evidence has been presented to show that drugs, toxic states, periods of anoxemia and myocardial ischemia, the presence of various catabolites, as well as changes in rate, affect the Ta wave both in normal sinus rhythm and during the arrhythmias.



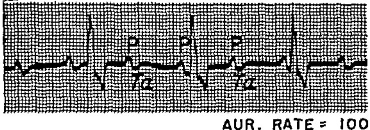


Figure 152 Auriculo-ventricular block produced by cutting Bundle of His in a dog. Lead 2. A shows 2.1 heart block; B after administration of digitalis. Note prominent Ta wave which results from administration of digitalis

mias. These observations indicate that the term "F" wave, intended to specifically designate the auricular deflection of flutter, should be discarded.

By the same methods used to study the P′ wave, the remaining portion of the flutter undulation is believed to consist of the Ta wave. This wave of regression or repolarization is made visible and larger than normal by the combination of two events: (1) the increase in rate; and (2) the progressive prolongation of the P′-R interval and the occurrence of auriculo-ventricular block. As in the normal electrocardiogram, the Ta wave in flutter and tachycardia is commonly inscribed in a direction opposite that of the wave of accession (P or P′ wave).

The effect on the electrocardiogram of a change in the position of the heart as reflected in the several leads has been described. Such positional changes may so alter the configuration of the auricular deflections in some leads that accurate diagnosis becomes difficult. It is thus obvious that, clinically, such positional changes of the heart as may be brought about by abnormal rotation, operation on the chest, trauma or other abnormal states may so affect the contour of the auricular deflections as to necessitate the use of special auricular leads as diagnostic aids.

The relationship of direct leads in animals to

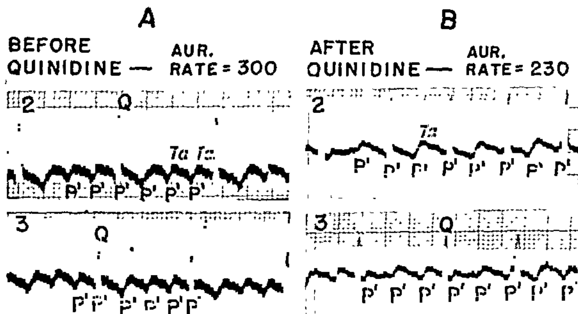


Figure 153. Effect of quinidine on experimental flutter. Leads 2 and 3

(A) Auricular rate is 300 beats per minute; no quinidine has been given

(B) After the administration of quinidine, the flutter is still present, the auricular rate has slowed to 230 beats per minute and the appearance of the flutter wave has changed because of the widening of the flutter wave

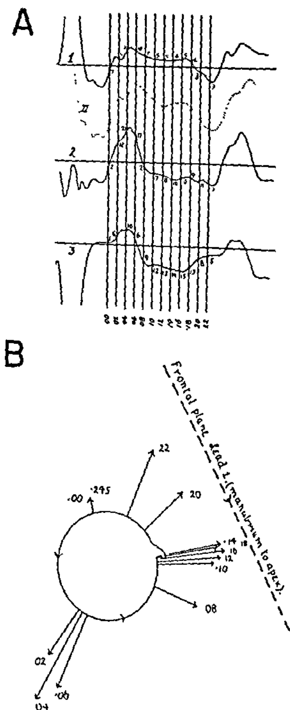


Figure 154 (A) Leads 1, 2 and 3 in frontal plane recorded by Lewis in patient with flutter Standard lead 2 is superimposed between frontal plane leads 1 and 2. The vertical lines are drawn at 0 02 second intervals
(B) Momentary atrial electrical axes in frontal plane calculated at 0 02 second intervals from electrocardiograms shown in A The calculations indicate ... wise movement ... On the b... of a circ... (From Sir Thomas Lewis's *Mechanism of the Heart Beat*, courtesy Shaw & Sons, Ltd, London)

oppositely directed Ta (repolarization) wave (Chapter VII), Lewis' conclusions are necessarily erroneous. The only part of the auricular deflection which reflects the activation of the auricle in flutter is the P′ component. As Lewis was not aware of this distinction, it would seem necessary to recalculate the momentary atrial electrical axes in his patient (Figure 155). Parallel observations made in one of our patients are shown in (Figure 157).

The instance of flutter studied by Lewis is of the common type. When the P′ and Ta waves in the electrocardiograms from his patient are delineated, the momentary atrial axes of the P′ waves in the standard limb leads do not indicate the existence of a circus movement. Quite the contrary, the excitation wave starts at the lower end of the auricles and goes forward and upward in a simple and direct manner (Figure 156). The momentary atrial axes in our own patient (Figure 157) with the common type of flutter were similar to those in Lewis' patient; the axes of the P′ waves were directed upward, those of the Ta waves downward.

The circus movement theory of flutter thus was based on a false belief that the momentary electrical axis of the flutter undulation revolves through 360 degrees. Had Lewis recognized the specific component parts of the flutter wave, and the fact that the Ta wave of repolarization is inscribed in a direction opposite that of the P′ wave of depolarization, presumably he would not have concluded that the flutter impulse travels in a circular pathway. It was this same reasoning from a fallacious assumption which led Decherd, Ruskin and Herrmann,[135] and Grishman and co-workers [219] to arrive at similar invalid conclusions.

THE ESOPHAGEAL LEAD ELECTROCARDIOGRAM OF SPONTANEOUS AURICULAR FLUTTER IN MAN

In the present study the nature of flutter in man was investigated by means of esophageal leads recorded in 10 patients. Analysis of the

CHAPTER VIII

Electrocardiographic Observations of Spontaneous Auricular Flutter and Auricular Paroxysmal Tachycardia In Man

CINEMATOGRAPHIC and electrocardiographic studies of experimentally produced auricular flutter and tachycardia in the dog (Chapters III, IV, V, VI, VII) have shown these arrhythmias to consist of rapidly recurring waves arising from a single ectopic focus and spreading in all available directions simultaneously. In no experiment was the phenomenon of re-entry observed. Regardless of their clarity and accuracy, however, results of animal experiments cannot be applied directly to man, such observations serve merely as guides for analogous clinical investigations. Consequently, a series of observations was made to determine whether or not the clinical forms of auricular flutter and auricular paroxysmal tachycardia exhibit the same electrical features found in the experimentally produced arrhythmias.

COURSE OF THE EXCITATION WAVE OF CLINICAL AURICULAR FLUTTER

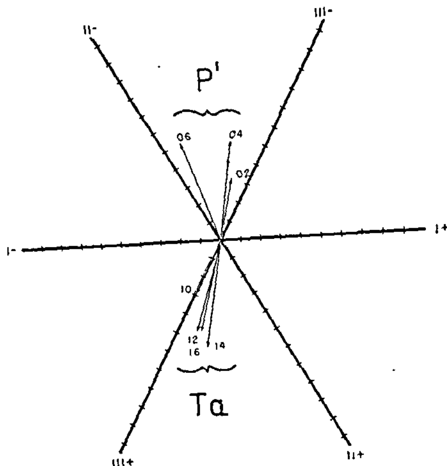
The nature of flutter in man heretofore has been investigated by essentially only one method, namely, a detailed examination of the momentary atrial electrical axis of the auricular deflection. Lewis [372] and co-workers recorded three simultaneous leads arranged in frontal, sagittal and horizontal planes of the body of a patient with auricular flutter (Figure 154) The momentary electrical axes of the auricles were analyzed at 0.02 second intervals in each of these planes, and plotted graphically to determine the course of the electrical events during the cycle of auricular flutter From Lewis' study the conclusion was drawn "that this (electrical) axis revolves during the progress of each auricular cycle through 360 degrees. This revolution of the axis gives us, so we believe, incontestable evidence that the movement of the excitation wave throughout the auricle as a whole *is controlled by a re-entrant movement around* a circle or an ellipse." [372]

Decherd, Ruskin and Herrmann [135] repeated Lewis' experiment but modified his technique in the following manner. The electrode connections were varied slightly since only two simultaneous leads were recorded. By an ingenious photographic method these workers were able to visualize on a three dimensional scheme what they considered the pathway of the excitation wave. Their conclusions were largely in agreement with those of Lewis. Grishman, Kroop, Jaffe and Steinberg [219] also arrived at a similar conclusion.

Although Lewis considered his results unequivocal, Scherf, [541] Brams and Katz, [64] Wilson, [656] and others, have pointed out that the muscular mass contained in the narrow mother pathway of the hypothetical circuit is too small to produce the potential recorded as the flutter wave by the electrocardiograph. Other criticisms of Lewis' method for determining the course of the flutter wave in man will be presented in this chapter

OBSERVATION I: RECALCULATION OF THE MOMENTARY ATRIAL ELECTRICAL AXES IN A CASE OF AURICULAR FLUTTER ANALYZED BY LEWIS

If the flutter wave consists of two components, a P' (depolarization) wave, and an



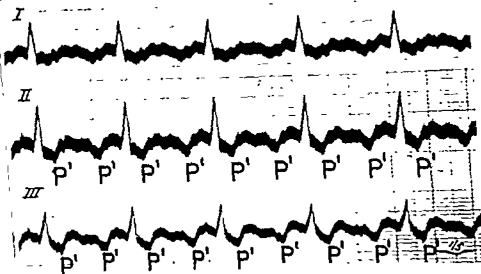


Figure 156 (A) Calculation of the momentary atrial electrical axes from standard limb lead The axes of the P' waves recorded from 0 02 through 0 06 second all point in the same general upward (cephalic) direction between III— and II— in the reference axes The axes of the Ta waves are directed downward (caudad) through points 0.10 to 0 14 and are between III+ and II+. The momentary atrial electrical axes of depolarization do not indicate the existence of a circus movement; indeed they cover a range of less than 60° instead of the 360° of the hypothetical circuit

(B) The standard limb leads 1, 2 and 3 from which the above data were obtained The momentary axes were calculated at 0 02 second intervals from the onset of a P' wave through one auricular cycle. (From Sir Thomas Lewis's *Mechanism of the Heart Beat*, Courtesy Shaw & Sons, Ltd , London)

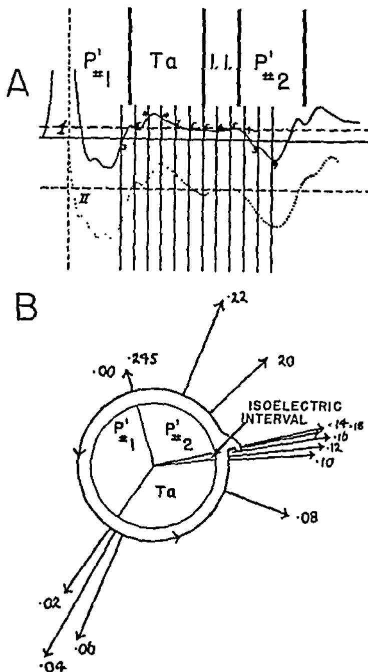


Figure 155. Further recalculation of Lewis's data on the momentary atrial electrical axes in flutter. Modification of Lewis's preceding figure (Figure 154)

(A) Lead 1 in frontal plane above, standard lead 2 below (dotted tracing). Examination of the electrocardiogram demonstrates that Lewis's measurements included the portion of a P' wave following a ventricular complex (P' #1), the succeeding Ta wave, an isoelectric period, and a portion of the succeeding P' wave (P' #2) The deflection which Lewis assumed to represent a single wave of depolarization actually started (0 00 point) at the middle of the succeeding wave of depolarization

(B) Diagram of Lewis's circus pathway, modified to show each of the electrocardiographic components We have delineated the part of the circle corresponding to each wave in the tracing. The end of the first depolarization wave (P' #1) occurred near the 0 00 point Auricular repolarization then began and the axis of 0.02 became oppositely directed to that of 0 00, the second third of the circle (0.02 - 1.11) thus represents a repolarization wave (Ta wave) and is followed by a brief isoelectric period (0 12 - 0 17) The second depolarization wave (P' #2) was incomplete, it accounts for the remaining portion of the circle *When the component parts of the flutter wave* are thus analyzed, the inaccuracy of Lewis's calculations is obvious Lewis, who did not distinguish the P' and Ta components of the flutter deflection, based his measurements on parts of two depolarization waves, one repolarization wave and one isoelectric period rather than on a single depolarization wave

large, easily discernible, auricular intrinsicoid deflections from these leads provides a method of accurately charting the course of the flutter excitation wave. In three of the patients the precordial leads also exhibited large, readily recognized P' waves by means of which the course of the flutter wave could be charted over the chest. Esophageal leads of flutter have been studied in the past by Brown[74] and others.[166, 317]

Anatomic Relationship of Esophagus to Auricles: To fully understand the significance of the esophageal tracings presented in this chapter, the reader should be familiar with the anatomic relationship of the *esophagus to the two* auricles and the interauricular septum. In Figure 158, depicting a posterior view of the mediastinum dissected from a cadaver with a normal heart, the relationship of the esophagus to the right and left auricles is shown. The esophagus has been removed, the auricles *opened, and the location of the interauricular* septum and each auricle in relation to the esophagus is seen. At the mid-auricular level (30.0 to 37.0 centimeters from lips), the esophagus is in close association with the left auricle and *is directly under the septum At the* level of the extreme caudal end of the auricles (37.5 to 40.0 centimeters from lips), the esophagus appears to be in more intimate contact with the right than with the left auricle. At the level of the cephalic end of the auricles (20.0 to 30.0 centimeters from lips), the esophagus *is* in con*tact with only the left auricle, since* the right auricle drops away anteriorly. The esophageal leads, then, bear the same general *relationship* to the auricles with reference to the interauricular septum as the precordial leads bear to the ventricles with reference to the interventricular septum. It is thus apparent that throughout the



Figure 158 Photographs of auricles and esophagus, posterior view, showing the relationship of the esophagus to the auricles Esophagus has been resected and each auricle opened The area formerly occupied by the esophagus is delineated by a dotted line When seen in posterior view, the esophagus lies directly over the midportion of the interauricular septum and over the cephalic portion of the left auricle At the lower part of its course, just above the diaphragm, the esophagus appears to be closer to the right auricle than to the left

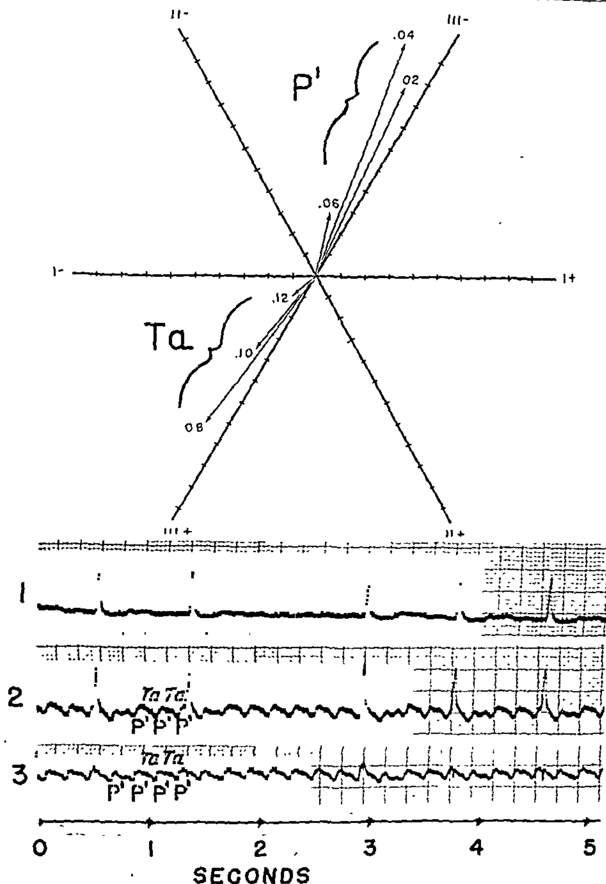


Figure 157. Momentary atrial electrical axes from a patient with heart block and the common type of auricular flutter. The momentary atrial electrical axes of the P′ and Ta waves were calculated. As in Figure 156, the P′ wave rotates in a cephalic direction through a distance of 20° between II− and III−. The Ta wave rotates only about 30° and travels in the general direction of III+ (a caudal direction). The electrocardiograms were enlarged 10 times in order to calculate these axes



Figure 158 Photographs of auricles and esophagus, posterior view, showing the relationship of the esophagus to the auricles Esophagus has been resected and each auricle opened The area formerly occupied by the esophagus is delineated by a dotted line When seen in posterior view the esophagus lies directly over the midportion of the interauricular septum and over the cephalic portion of the left auricle At the lower part of its course, just above the diaphragm, the esophagus appears to be closer to the right auricle than to the left

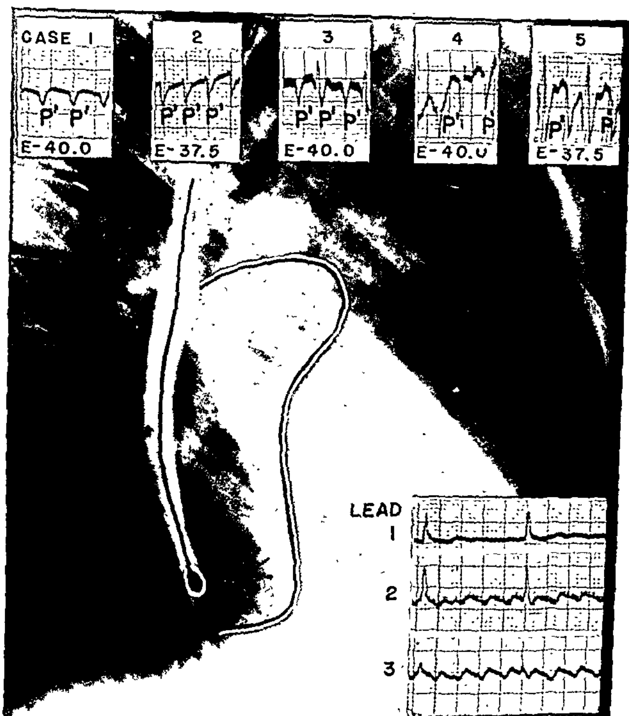


Figure 159 Tracings from five patients recorded from electrodes in the esophagus approximately 37.5 to 40 cm. from the lips This portion of the esophagus lies directly posterior to the caudal region of the auricles. The patients exhibit the common type of flutter with inverted P' waves in leads 2 and 3 as exemplified in the standard limb lead electrocardiogram shown below Note that in each instance the tracing from this low auricular level displays a negative deflection, indicating that the impulse starts at the caudal end of the auricles

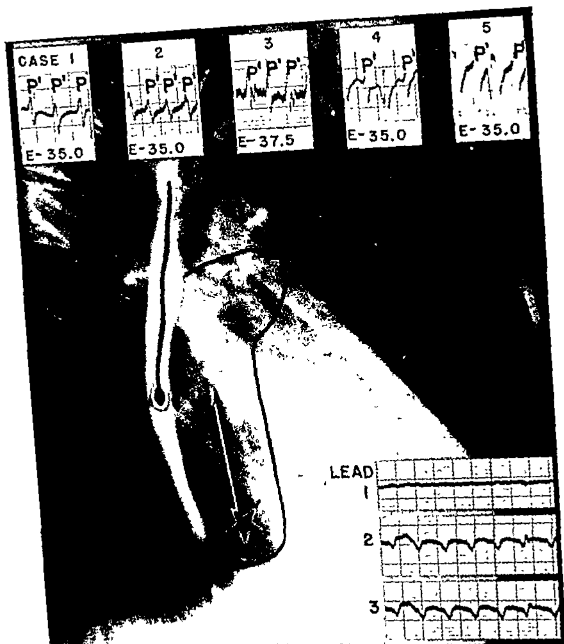


Figure 160 Esophageal electrocardiograms of the common type of flutter recorded from electrodes 35 to 37 5 cm from the lips (This portion of the esophagus lies immediately posterior to the mid-portion of the auricles) Cases 1 through 5 same as in Figure 159 In each tracing from this mid-auricular level a distinct positive deflection precedes the negative deflection indicating that the impulse started at the caudal end of the auricle, passed the electrode and traveled toward the cephalic end of the auricle. Below is standard limb lead electrocardiogram from one of these patients

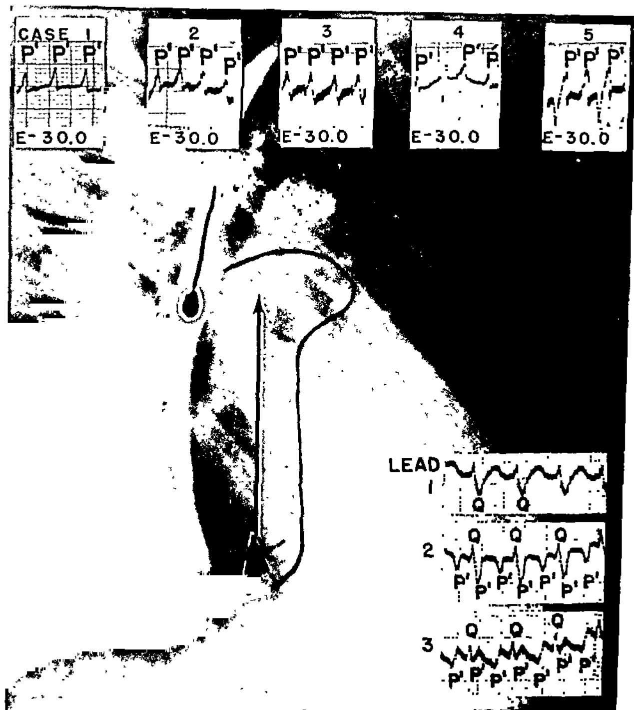


Figure 161. Esophageal tracings of the common type of flutter recorded from electrodes approximately 30 cm. from the lips This portion of the esophagus lies directly under (corresponds to) the cephalic end of the auricles Same patients as in Figures 159 and 160 In each instance the tracings from this high auricular level displays a primary positive deflection, indicating that the impulse travels toward the electrode during most of its course The impulse of the common type of flutter thus *starts at the caudal end of the auricle* (Figure 159), traverses the mid-auricular region (Figure 160) and reaches the top of the auricle (Figure 161) The standard limb lead electrocardiogram shown below is from one of these patients

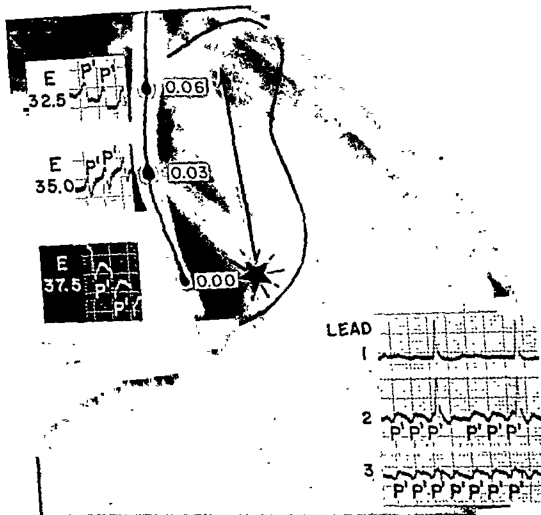


Figure 162 Simultaneous esophageal leads from three auricular levels (E 32 5, E 35 0 E 37 5) in a patient with the common type of flutter (rate 300 per minute) A pure negative auricular deflection is recorded from the electrode over the caudal end of the auricle (E 37.5). In the tracings from higher levels the negative deflection is preceded by a positive deflection; the tracing recorded over the cephalic end of the auricle is completely positive The intrinsic deflections at successively higher levels are recorded at progressively later periods Thus the ectopic focus is at the caudal end of the auricles and the impulse travels in a caudocephalic direction. Standard limb leads are shown at lower right.

greater part of its course adjacent to the auricular structures, *the esophagus must receive impulses* from both right and left auricles. Esophageal tracings recorded from an electrode approximately 35.0 to 40.0 centimeters from the lips reveal the electrical events occurring at the caudal end of the auricles; electrodes 32.5 to 37.5 centimeters from the lips register events in the mid-portion (body proper) of auricles; electrodes approximately 30.0 to 35.0 centimenters from the lips record events in the cephalic end of the auricles.

Form of the Auricular Deflections in Esophageal Lead Electrocardiograms: The interpretation of esophageal lead electrocardiograms from man is based upon the same principles of unipolar electrocardiography applied in our analysis of direct auricular lead tracings from animals (Chapters I, II, III, IV and VI).

According to these concepts, the exploring electrode faithfully records electrical events in the portion of the auricle overlying it; the potential from the indifferent electrode is minimal.[266, 636] The polarity of the electrodes is so arranged that an impulse traveling away from the electrode (i.e., the electrode faces the tail of the wave) registers a negative deflection, an impulse directed toward the electrode (electrode faces the head of the wave) registers a positive deflection. If an impulse starts at one end of a linear strip of muscle and travels to the other end, the electrode at the center of the strip registers a positive deflection until the impulse passes beneath it; at this moment the deflection becomes negative (Figure 5, Chapter I). The sharp downward deflection recorded as the impulse passes beneath the electrode is known as the intrinsic deflection.

In the following studies, the esophageal electrode is the exploring electrode; electrical activity in the adjacent parts of the auricle is recorded from it.

The esophageal lead electrocardiograms from seven of the patients with flutter described in this chapter are almost identical in appearance. The P′ deflection is deeply inverted and the Ta wave is upright in leads 2, 3 and AVF. These cases represent the common type of flutter characterized by inverted P′ waves in leads 2, 3 and AVF (Chapter IX) and are considered as a group. The eighth and ninth cases represent an uncommon variety of flutter in which the P′ wave is upright in leads 1 and 2; these records are considered separately. Lastly, the rare occurrence of both the common and uncommon types of flutter in the same patient is described.

Observation 2: Configuration of the Auricular Deflections in Esophageal Lead Electrocardiograms of the Common Type of Clinical Auricular Flutter

A pure negative auricular deflection was always recorded from the esophageal electrode at or below the caudal level of the auricles in each of the seven patients exhibiting the common type of auricular flutter (electrocardiograms of five of the seven cases are shown in Figure 159). In esophageal tracings from higher levels adjacent to the body of the auricles, the negative wave was preceded by a small positive wave. In tracings from successively higher levels, the positive wave was progressively larger (Figure 160). In esophageal lead tracings recorded at or above the cephalic level of the auricles the P′ wave was entirely upright; the inverted portion had completely disappeared (Figure 161).

Figure 162 shows three simultaneous esophageal leads from different levels in a patient with the common type of flutter

Significance of the Pure Negative Deflection: In the light of the principles of unipolar electrocardiography discussed above, a pure negative deflection in the esophageal lead from the low auricular level indicates that the impulse arises from an ectopic focus at or near this region, and travels away from the point of origin. In the group of patients exhibiting the common type of flutter, therefore, the caudal end of the auricle must be the site of the ectopic focus.

During our experimental study of flutter in dogs, the caudal end of the auricle was by far the most susceptible area for the production of flutter. Thus in both man and dog there appears

to be predilection for the development of a flutter focus in the caudal end of the auricle.

Significance of the Biphasic Deflection: When the electrode is raised from the esophageal region adjacent to the caudal end of the auricle to a position behind the auricular body (Figure 162), the negative deflection in the tracing is preceded by a positive wave. The deflections in tracings from this region are essentially biphasic in configuration. As pointed out above, the upright deflection indicates that the impulse is moving toward the electrode from its site of origin in the caudal part of the auricle. After the impulse passes beneath the electrode and continues in a cephalic direction, the electrode faces the tail of the impulse and a negative deflection is recorded. The intrinsicoid deflection occurs at the instant the impulse passes beneath the electrode.

Significance of the Positive Deflection: Tracings from electrodes at the cephalic level of the auricles (Figure 162) consist of pure positive deflections; such waves are in opposite direction to those obtained at the caudal level of the auricles. A purely positive deflection shows that the impulse at all times is traveling toward the electrode and never away from it. Since no [illegible]

[illegible] at the cephalic region of the auricle. If the impulse continued to travel (as it necessarily would if a circus movement were present) it would soon be moving away from the electrode (caudally) and a *negative deflection* would be recorded.

The above analysis of the shapes of auricular deflections in esophageal lead electrocardiograms from seven patients warrants the following conclusions. The wave of depolarization in the *common type of flutter* in man originates [illegible]

[illegible] region of origin of spontaneous flutter in man is the same as the most susceptible region for production of experimental flutter in dogs. The course of the excitation wave from this site of origin in spontaneous flutter *in man* is similar to the course of the contraction and excitation waves of experimentally produced flutter in dogs (Chapters V and VI).

Observation 3: Theoretical Form of the Esophageal Lead Electrocardiogram of the Common Type of Auricular Flutter in "Circus Movement"

The original concept of the circus movement postulates that once the flutter rhythm is initiated the movement of the cardiac impulse continues in an unbroken circuit without beginning or end. Therefore, the tracings from electrodes at all points on the main pathway should be identical. In Chapters V and VI the fact was established that this does not hold true in the dog; corresponding observations reported in this chapter demonstrate that it is also untrue in man. Definite and predictable variations in the auricular deflections regularly occur in direct auricular leads in the dog and in esophageal leads in man. Similar variations are also seen in Lewis' records from dogs and in tracings from esophageal leads in man recorded by other workers.

In order to meet this objection to the original circus movement theory, Wilson[656] suggested the possibility that auricular flutter consists of a circus movement which begins *anew* at the same ectopic focus with each circuit; in other words, a separate impulse issues from the original focus at the onset of each cardiac cycle and travels along a circus pathway, terminating upon completing the circuit. Barker, Wilson and Johnson[23] believed a similar mechanism might be present in tachycardia. If either Lewis' original circus movement theory or Wilson's suggested modification thereof were correct, the following theoretical assumptions concerning the form of the auricular deflection in esophageal lead electrocardiograms of the common type of flutter should prove true.

In tracings from an esophageal electrode placed near the focus at the caudal end of the auricle, a negative deflection would be inscribed until the impulse reaches the cephalic end of the auricle (Figure 163). It must be remem-

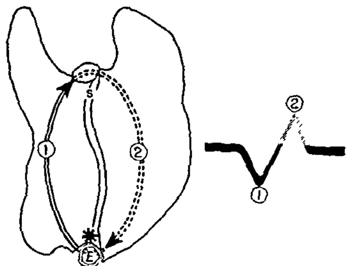


Figure 163. Hypothetical drawing, showing form of the auricular deflection in an esophageal electrocardiogram from the level of the caudal end of the auricles as it would appear if a circus movement were present. The ectopic focus as represented by a star is at the caudal end of the auricles. Recording electrode is represented by "E."

As the impulse traveled toward the cephalic end a negative wave would be recorded (1). As the impulse returned to complete the hypothetical circuit, it would travel toward the electrode and would inscribe a positive deflection (2). Thus, the resulting wave would be biphasic. In seven instances of the common type of flutter examined by esophageal leads, the tracings from low auricular levels were purely negative, the positive wave (shaded) portion of the hypothetical deflection was never recorded Hence, the impulse did not return to the focus

bered that the electrical events in both auricles are recorded from the esophagus. Therefore, on the hypothetical return journey down the other auricle the oncoming wave would be faced by the electrode, a *positive deflection* should then appear and become progressively larger as the wave approaches the focus (Figure 163). *In fact, no such positive wave occurs,* the deflection recorded by the electrode at the level of the focus is completely negative. Hence, *the impulse does not return to the focus and no circus movement is present.*

With the electrode in mid-auricular position, *the impulse would register a positive deflection* on its journey from the caudal focus until it reached the level of the electrode (Figure 164). *A negative deflection would then be inscribed* as the impulse traveled from the level of the electrode to the cephalic end of the auricle, completing the first half of the hypothetical circuit (*Figure 164*). On its return journey, the impulse would turn downward; a positive deflection would be recorded until it reached the level of the electrode, then a negative deflection as it continued to travel back to the focus. If the flutter impulse pursued a circus pathway, therefore, each auricular complex in esophageal electrocardiograms from the mid-auricular level would consist of a series of two positive-negative waves (positive-negative, positive-negative). The second "positive-negative" does not occur. Hence, there is no return journey.

With the electrode at the cephalic level of the auricle, the impulse traveling upward from the focus would register a positive deflection until it reached the level of the electrode (Figure 165). If the impulse then returned to the focus, it would register a deep negative deflection as it traveled down the auricle away from the electrode. No negative deflection is recorded from electrodes at the cephalic level.

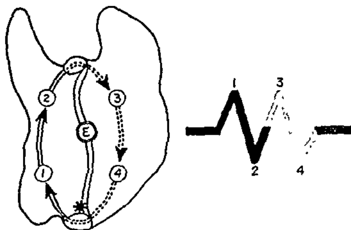


Figure 164 Theoretical form of the esophageal electrocardiogram from an electrode "E" at the mid-auricular level if circus movement were present Focus is at the caudal end of the auricles. As the impulse traveled in a cephalic direction from the focus as represented by star, a positive deflection would be recorded (1), after the impulse had passed the electrode it would register a negative wave (2). *As the* impulse returned to complete the hypothetical circuit these events would repeat themselves, *a second positive wave* (3) would appear as the impulse approached the electrode *on the return journey*, followed by a negative wave (4) as the impulse again passed the electrode and neared the site of origin Only the first positive and negative waves are, in fact, recorded, the second positive and negative waves (shaded) do not occur Therefore, the impulse does not return to the focus

Therefore, the impulse must terminate at the cephalic end of the auricles, no circus movement is present.

In summary, in the common type of flutter the impulse starts at the caudal end of the auricle, reaches the top of the auricle, and terminates In the seven instances of spontaneous flutter in man examined by means of esophageal leads as described above, no evidence was found to suggest that this movement represents the first half of a circle. Esophageal leads of flutter recorded by others likewise exhibit the simple waves observed in these seven instances. To our knowledge, none of the complex deflections which would have to be present if a circus movement existed has ever been described.

The course of the impulse in the common type of flutter in man as traced with esophageal leads from the caudal, cephalic and mid-auricular levels is identical with the course of the impulse in a simple muscle strip activated at one end and traced with electrodes at each end and at the center (Figure 166) The simplicity of the course of the flutter wave in man is thus evident Like the excitation waves of normal sinus rhythm, auricular premature systole and paroxysmal tachycardia, the flutter wave pursues a linear path

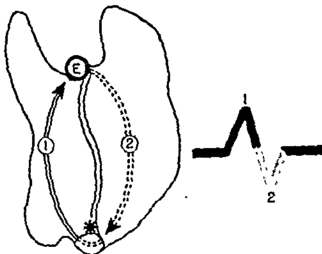


Figure 165 Theoretical representation of the auricular deflection from an electrode in the esophagus at the level of the extreme cephalic end of the auricles as it would appear if a circus movement were present. Flutter focus is at the caudal end of the auricle. As the impulse traveled from the focus to the electrode a positive wave (1) would be inscribed As the impulse then left the cephalic end of the auricle and returned to the caudal end a negative wave (2) would be recorded. In esophageal tracings from high auricular levels in all of seven patients studied, only the positive wave was found, the negative (shaded) portion was nonexistent. Therefore, the impulse travels from the caudal to the cephalic end of the auricle where it terminates, it does not return to the focus.

Observation 4 Time of Onset of the Intrinsicoid Deflections in the Esophageal Lead Electrocardiogram of the Common Type of Clinical Auricular Flutter

In six patients exhibiting the common type of auricular flutter, the excitation wave was analyzed by determining differences in the time of onset of the intrinsicoid deflections from electrodes in varying esophageal positions. In two subjects four esophageal leads were recorded simultaneously In the remaining four patients three esophageal leads were recorded from various esophageal levels simultaneously with lead AVF, lead AVF was used as a reference point for purposes of timing.

As reported above, a pure negative deflection is recorded in esophageal tracings from the caudal level of the auricles, since this intrinsic deflection represents the onset of the excitation wave, it occurs at 0.00 second. The intrinsicoid deflections occur progressively later in the records inscribed from successively higher auricular levels (Figures 162 and 167). The intrinsicoid deflection from the cephalic level of the auricle appears 0.04 to 0.06 second later than that from the caudal level of the auricle. The impulse therefore must travel from the ectopic focus at the caudal end of the auricle in a caudocehpalic direction until it reaches the cephalic end.

This conclusion regarding the course of the flutter wave as determined by timing the onset of the intrinsicoid deflections in simultaneously recorded esophageal lead tracings is identical with that derived from an examination of the configurations of the auricular deflect[illegible]

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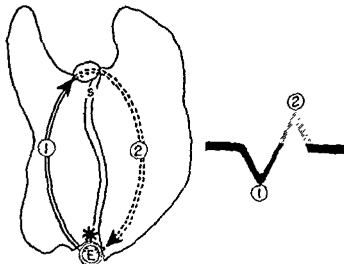


Figure 163. Hypothetical drawing, showing form of the *auricular deflection* in an esophageal electrocardiogram from the level of the caudal end of the auricles as it would appear if a circus movement were present. The ectopic focus as represented by a star is at the caudal end of the auricles. Recording electrode is represented by "E."

As the impulse traveled toward the cephalic end a negative wave would be recorded (1). As the impulse returned to complete the hypothetical circuit, it would travel toward the electrode and would inscribe a *positive deflection* (2). Thus, *the resulting wave would be biphasic.* In seven instances of the common type of flutter examined by esophageal leads, the tracings from *low auricular levels* were purely negative, the positive wave (shaded) portion of the hypothetical deflection was never recorded. Hence, the impulse did not return to the focus.

bered that the electrical events in both auricles are recorded from the esophagus. Therefore, on the hypothetical return journey down the other auricle the oncoming wave would be faced by the electrode; a positive deflection should then appear and become progressively larger as the wave approaches the focus (Figure 163). In fact, no such positive wave occurs; the deflection recorded by the electrode at the level of the focus is completely negative. Hence, *the impulse does not return to the focus* and no circus movement is present.

With the electrode in mid-auricular position, the impulse would register a positive deflection on its journey from the caudal focus until it reached the level of the electrode (Figure 164). A negative deflection would then be inscribed as the impulse traveled from the level of the electrode to the cephalic end of the auricle, completing the first half of the hypothetical circuit (Figure 164). On its return journey, the impulse would turn downward; a positive deflection would be recorded until it reached the level of the electrode, then a negative deflection as it continued to travel back to the focus. If the flutter impulse pursued a *circus pathway,* therefore, each auricular complex in esophageal *electrocardiograms from the mid-auricular level* would consist of a series of two positive-negative waves (positive-negative, positive-negative). The second "positive-negative" does not occur. Hence, there is no return journey.

With the electrode at the cephalic level of the auricle, the impulse traveling upward from the focus would register a *positive deflection* until it reached the level of the electrode (*Figure 165*). *If the impulse then returned to* the focus, *it* would register a deep negative deflection as *it* traveled down the auricle away from the electrode. No negative deflection is recorded from electrodes at the cephalic level.

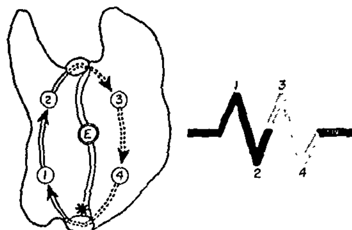


Figure 164. Theoretical form of the esophageal electrocardiogram *from an electrode "E" at the mid-auricular level* if circus movement were present. Focus is at the caudal end of the auricles. As the impulse traveled in a cephalic direction from the focus as represented by star, a positive deflection would be recorded (1), after the impulse had passed the electrode it would register a negative wave (2). As the impulse returned to complete the hypothetical circuit these events would repeat themselves, a second positive wave (3) *would appear as the impulse approached the electrode* on the return journey, followed by a negative wave (4) as the impulse again passed the electrode and neared the site of origin. *Only* the first positive and negative waves are, in fact, recorded, the second positive and negative waves (shaded) do not occur. Therefore, the impulse does not return to the focus.

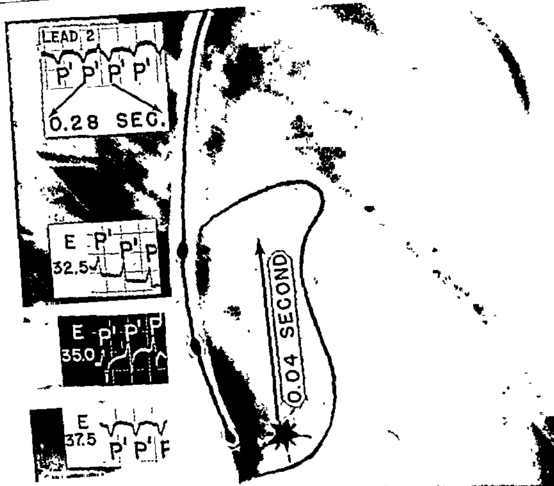


Figure 167 The direction of spread of the flutter wave as determined by comparing the times of onset of the ... electroc ... in the ... intrinsic ... focus at the caudal end of the auricle, it is recorded at time of onset in the cycle 0 00 second The intrinsic deflections in the tracings from successively higher levels occur progressively later, the deflection from the top of the auricle is recorded approximately 0 04 second after that from the bottom. Thus, the impulse must start at the caudal end and travel in a caudocephalic direction This direction of progression of the flutter impulse occurs in all instances of flutter with inverted P' waves in leads 2, 3 and AVF (common type of flutter).

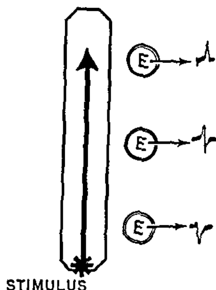
0 28 second. Hence, each cardiac cycle began 0.28 second instead of 08 second after the onset of its predecessor Indeed, only if the auricular rate were 750 per minute would the P'-P' interval equal the calculated .08 second, yet such a rate is far beyond the fibrillation threshold. Similar inconsistencies between the hypothetical and actual length of the P'-P' interval were found in the five other instances in which corresponding measurements were made. These findings are compatible with the existence of a circus movement only if the speed of the wave along one-half of the circuit (return journey) is several times slower than during the other half (forward journey) or if a delay occurs between the termination of one circuit and the beginning of the succeeding circuit. The cinematographic counterpart of these observations is detailed in Chapter V.

By simultaneously recording esophageal leads from electrodes separated by fixed known distances, the approximate time required for an excitation wave to travel a given distance in the auricle can be determined. This is done by

SPREAD OF IMPULSE

A
SIMPLE MUSCLE STRIP
(THEORETICAL)

B
HUMAN AURICLES WITH
COMMON TYPE OF FLUTTER
(ACTUAL)



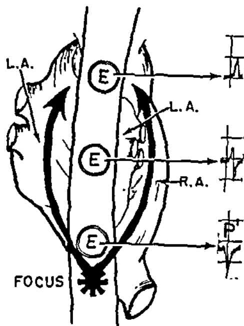


Figure 166. (A) Demonstration of course of electrical impulse in a simple muscle strip Electrodes (E) are at either end and in the center of the strip, the stimulus is applied at the lower end (*). *To the right of the muscle strip are depicted the shapes of the deflections recorded from each of the three electrodes.*

(B) Three esophageal leads from a patient with flutter Electrodes (E) are at low, mid and high auricular levels The deflection from the low level is negative, that from mid level is biphasic, that from the high level is purely positive. The forms of the deflections derived from esophageal leads in flutter in man are identical with those in a single muscle strip.

The impulse in the common type of flutter in man is a simple wave which starts at the caudal end *of the auricle and travels upward to the cephalic end in exactly the same manner* as observed in the animal experiments reported in Chapters V and VI

flutter originates low in the auricles and travels in a caudocephalic direction.

Observation 5: Speed of the Excitation Wave of the Common Type of Auricular Flutter in Man

Lewis' theoretical circus pathway covers the cephalocaudal length of the auricles plus the caudocephalic length of the auricles. If the flutter impulse traveled in an unbroken circuit at a constant rate, as postulated by Lewis, the interval between the onset of succeeding cardiac cycles (P'-P' interval) would represent the length of time in which the impulse completes each circuit, or twice the time in which it traverses the cephalocaudal length of the auricles. As described above, in seven instances of *the common type of flutter,* the impulse was found to traverse the caudocephalic length of the auricles in approximately 0.04 to 0.06 second (Observation 4). In none of these instances did the P'-P' interval in the electrocardiogram approximate 0.08 to 0.12 second, as it would if Lewis' assumptions were valid. For example, approximately 0 04 second was required for the impulse, to traverse the first half of the circus pathway (caudocephalic length of the auricle) in the instance of *flutter illustrated in Figure* 168. In this instance the auricular rate was 214 beats per minute and the P'-P' interval was

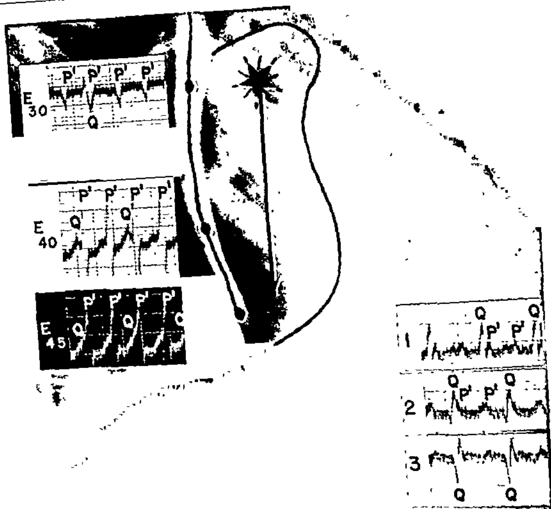


Figure 169 Esophageal electrocardiograms of an uncommon type of flutter, rate 215 (focus at cephalic end of auricle). Limb leads 1, 2 and 3 below are characteristic of a 2 1 flutter with upright P' waves in leads 1 and 2 The esophageal electrocardiogram from the electrode over the cephalic end of the auricle (E 30) is completely negative In tracings from successively lower levels an upward deflection appears and becomes progressively larger, at E 45 the deflection is completely positive.

In this uncommon type of flutter the impulse travels from the ectopic focus at the cephalic end toward the caudal end of the auricles. The course of the impulse is thus in the opposite direction to that found in the common type of flutter in which the focus is at the caudal end of the auricles and the impulse travels in a caudocephalic direction The P' waves in standard limb leads in the two types are also oppositely directed (Courtesy Charles D Enselberg, M D)

focus is at the caudal end of the auricle, in the uncommon type it is at the cephalic end. Neither type affords evidence in support of the circus movement theory.

Observation 7: Electrocardiographic Features of a Case Exhibiting Both Common and Uncommon Types of Auricular Flutter

The above studies of esophageal lead electrocardiograms of flutter have been concerned mainly with the elucidation of the mechanism of this arrhythmia by following the course of the impulse from a *single* ectopic focus. In the following pages an instance of flutter will be considered in which the impulse was found to arise at different times from *two* different foci *in the same patient.** On some occasions the esophageal lead electrocardiogram revealed the

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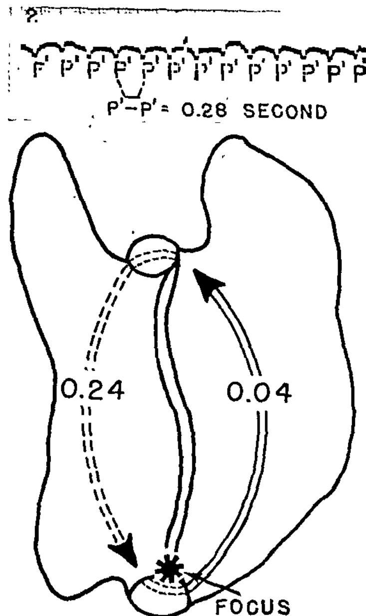


Figure 168 Demonstration, by timing the onset of the intrinsic deflections, that a circus movement in flutter is unlikely. In this patient the flutter impulse travels from the caudal to the cephalic end of the auricles in 0.04 second (see Figure 167) If the impulse continued along the theoretical circus pathway at the same speed it would return to the focus approximately 0.08 second after its departure The P'-P' interval actually is 0.28 second, hence, the succeeding impulse left the focus more than three times later than would be expected if a circus movement were present

dividing the distance between any two esophageal electrodes by the difference in the time at which the intrinsic deflections are registered by the electrodes. For example, if the electrodes are 5 centimeters apart and the intrinsic deflection from one is recorded 0.01 second before that from the other, the impulse traveled between the two electrodes at a rate of 5 centimeters per .01 second or 500 centimeters per second. This method was used to compare the speed of the excitation waves of normal sinus rhythm and auricular flutter in man. In a subject with normal sinus rhythm the speed of the impulse was approximately 500 centimeters per second; in a patient with the common type of flutter it was roughly 125 centimeters per second. Parallel observations on the difference in speed of the contraction waves of normal sinus rhythm and auricular flutter in animals were made cinematographically (Chapters I and V). This phenomenon was first observed by Lewis.[372]

Observation 6: The Esophageal Lead Electrocardiogram of the Uncommon Type of Clinical Auricular Flutter

Two of the instances of flutter included in this study exhibit upright P' waves in leads 1 and 2; such waves are directed exactly opposite those of the common type of flutter and appear in a relatively small percentage of patients (Chapter X). In this uncommon variety of flutter the auricular deflections in esophageal lead electrocardiograms from the *cephalic* level of the auricles are completely negative; those from the mid-auricular level are biphasic with an initial positive deflection followed by a negative phase, those from the lower auricular levels are completely upright (Figure 169). Thus the auricular deflections in esophageal leads, like those in the limb leads, are in exactly opposite direction to the deflections of the common type of flutter.

By comparing the times of onset of the intrisic deflections in various esophageal leads as described earlier, the impulse in these two instances of the uncommon type of flutter was found to travel the length of the auricle in a cephalocaudal direction. Thus the course of the impulse in this type of flutter is directly opposite that observed in the common type. In both types, the time required for the impulse to traverse the length of the auricle was approximately 0.04 second.

The basic difference between the common and uncommon types of flutter is the location of the ectopic focus. In the common type the

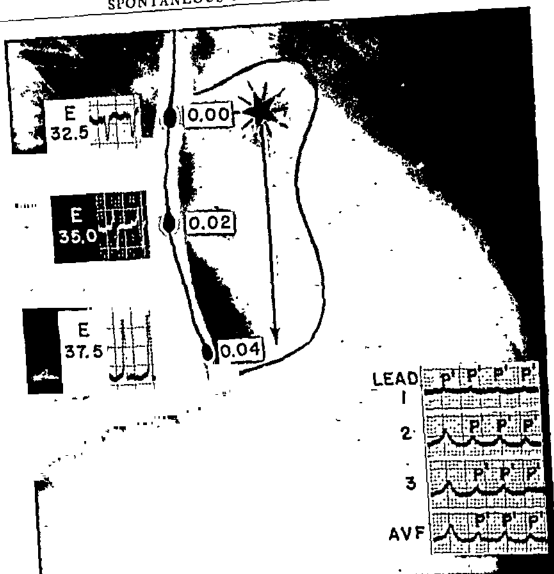


Figure 171. Esophageal and limb lead electrocardiograms recorded on March 15th from same patient as in Figure 170. Uncommon type of flutter is present The ectopic focus is at the cephalic end of the auricle (E 32 5) and the impulse travels in a cephalocaudal direction The P' waves are upright in leads 1, 2, 3 and AVF Auriculo-ventricular block is still present

ance of complete heart block. Since August 23, 1949, tracings have shown auricular flutter in addition to heart block.

When the patient was first examined in this laboratory on March 5, 1950, 9½ months after the onset of the diphtheria, an electrocardiogram exhibited the common type of flutter with inverted P' waves in leads 2, 3 and AVF; the auricular rate was 214 beats per minute (Figure 170). Esophageal leads taken on that day showed pure negative deflections from the caudal level of the auricle (E 37.5) and completely upright deflections from the cephalic level (E 30), indicating that the flutter was of the common type in which the impulse starts at the caudal end of the auricle and travels in a caudocephalic direction (Figure 170).

On March 15, the electrocardiogram presented a remarkable change. The auricular rate was still 214 beats per minute, but the P' waves had become upright in leads 2, 3 and AVF (Figure 171). In the esophageal leads pure negative deflections were obtained from the cephalic level of the auricle (E 32.5); the de-

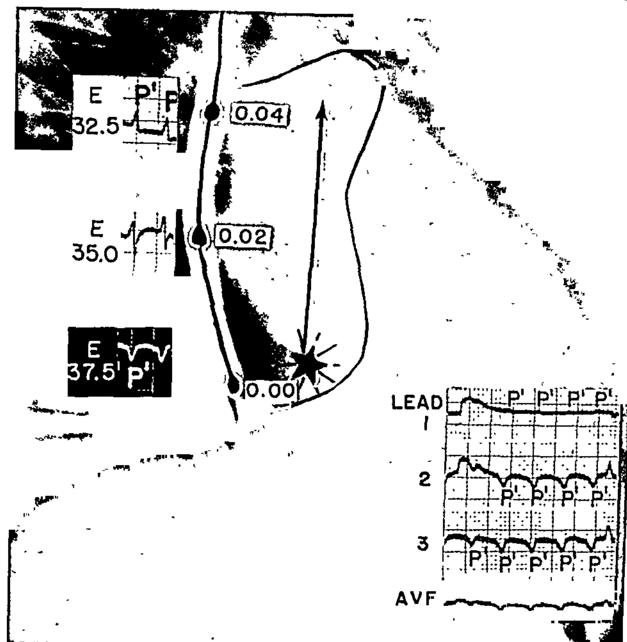


Figure 170 Tracings from unusual patient alternately displaying common and uncommon types of flutter. Esophageal and limb lead electrocardiograms recorded on March 5th, the common type of flutter, rate 214, is present The ectopic focus is at the caudal end of the auricles (E 37.5) and the impulse travels in a caudocephalic direction Note inversion of the P' wave in leads 2, 3 and AVF Auriculoventricular block is present

common type of flutter (from a focus at the caudal end of the auricle); on another occasion the uncommon type (from a focus at the cephalic end) was present. An additional feature of this case enhancing its value for study purposes was the simultaneous occurrence of complete auriculo-ventricular block as a result of which the auricular deflections could easily be seen.

The patient was a 37 year old female whose general health had been satisfactory until May 20, 1949, when she contracted diphtheria which responded to treatment with antibiotics and antitoxin. Five months later she experienced subjective cardiac symptoms for the first time. Congestive heart failure was diagnosed and proved highly refractory to treatment.

On May 27, 1949, during the first period of hospitalization, an electrocardiogram was normal. A tracing recorded three days later showed left bundle-branch block and a wandering pacemaker; this was soon followed by the appear-

flections from successively lower levels were progressively more upright and those from the caudal level (E 37.5) were completely positive. Obviously, the ectopic focus on this occasion was located at the cephalic end of the auricles and the course of the impulse was in a cephalo-caudal direction. Associated with this change were upright P′ waves in all three standard limb leads (Figure 171).

Another feature in this case is worthy of note. On two occasions (March 7 and March 17) extremely slow auricular rates were found, approximately 30 beats per minute. (Figure 172C). In tracings recorded on both these occasions P′ waves were present and were of the same two general types recorded during the episodes of flutter, namely, upright (uncommon type) (Figure 172A) and inverted (common type) (Figure 172B). Ta waves, which were clearly visible in the other tracings of flutter (most marked in the common type), were not inscribed during the slow-rate episodes. No subjective symptoms were experienced by the patient during the changes in rhythm.

In summary, the record of this patient is instructive from three standpoints: (1) The two most frequently encountered types of flutter were seen to occur in the same patient. (2) The two ectopic foci responsible for the two types of flutter were also active at slow auricular rates. It is evident that during the slow-rate intervals the foci at the caudal and cephalic ends alternated as cardiac pacemakers; during the rapid-rate (flutter) episodes one focus was more active than the other and dominated the auricular rhythm. (3) Ta waves were definitely present during the fast-rate (flutter) episodes but were absent when the rates were slow. This fact demonstrates the effect of auricular rate on the production of Ta waves in both the common and uncommon types of flutter.

THE PRECORDIAL LEAD ELECTROCARDIOGRAM OF SPONTANEOUS AURICULAR FLUTTER IN MAN

When large P′ waves with clearly discernible intrinsicoid deflections are recorded from multiple precordial leads, the time of onset of each intrinsic deflection can be determined and a map constructed which shows the course of the excitation wave. An area on the chest which yields a pure negative deflection must first be found. As explained above, this area corresponds to the ectopic focus; the impulse passes beneath it at 0.00 second. The times of onset of the intrinsic deflections obtained from the 0.00 point and from any other point on the chest are then compared by using a simultaneously recorded limb lead as a reference point. Thus the relative times at which the impulse passes beneath various points on the chest is determined.

Unfortunately, in most electrocardiograms from patients with flutter the intrinsicoid deflections in chest leads are either invisible or too indistinct to permit such study. In the present series only two patients exhibited clear intrinsicoid deflections in precordial leads; both displayed the common type of flutter, as evidenced by the sharp, completely negative deflections from the lower end of the esophagus. In the precordial electrocardiogram pure negative deflections were found in each instance in the lead from the lower end of the sternum (V_E). By measuring the times of onset of the intrinsicoid deflections in the multiple precordial leads and using a simultaneously recorded lead AVF or lead 3 as a reference point, the course of the impulse was mapped on the chest of each patient (Figure 173). From the ectopic focus (point 0.00) beneath the lower end of the sternum, the impulse traveled outward and upward *in all available directions*, reaching areas in the chest equidistant from the focus at approximately the same time.

Both patients had auricular rates of 300 beats per minute; hence the P′-P′ interval was 0.20 second. If the impulse traveled around the auricles along a circus pathway in 0.20 [illegible] clockwise direction) would be recorded at successively later moments until the impulse completed the circuit at 0.20 sec-

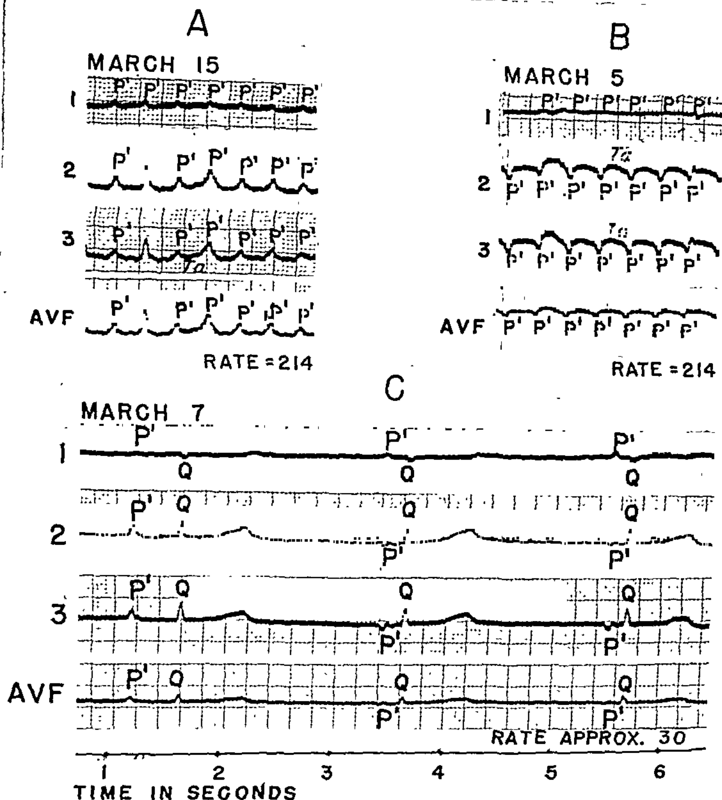


Figure 172. Standard limb leads and lead AVF from same patient as in Figures 170 and 171

(A) March 15th. Uncommon type of flutter with upright P' wave in leads 1, 2, 3 and AVF

(B) March 5th Common type of flutter with inverted P' waves in leads 2, 3 and AVF. Ta waves are present *and give tracing typical* undulatory appearance of flutter

(C) March 7th Auricular rate has slowed to about 30 beats per minute Two types of P' waves are present, upright and inverted, these are identical with those found when flutter was present (A and B) When the rate is slow as in C, the first P' wave is typical of the uncommon type of flutter and has the same configuration as in A, the second and third P' waves are characteristic of the common type of flutter and have the same configuration as in B Note that the Ta waves have disappeared

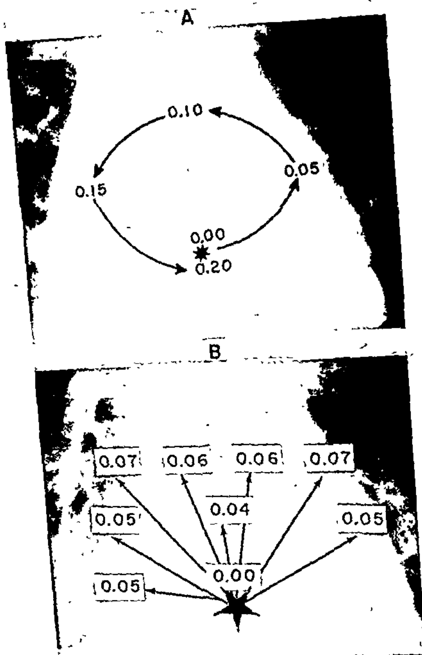


Figure 174 (A) Focus (*) is directly beneath the lower end of the sternum (point 0 00). In this patient the auricular rate was 300 beats per minute. An impulse following a ... around the ... clo... ch... ...of the flutter impulse, determined by timing the intrinsic deflections, is shown in Figure 173 The impulse radiates concentrically from the point of complete negativity (0 00), traveling in all available directions.

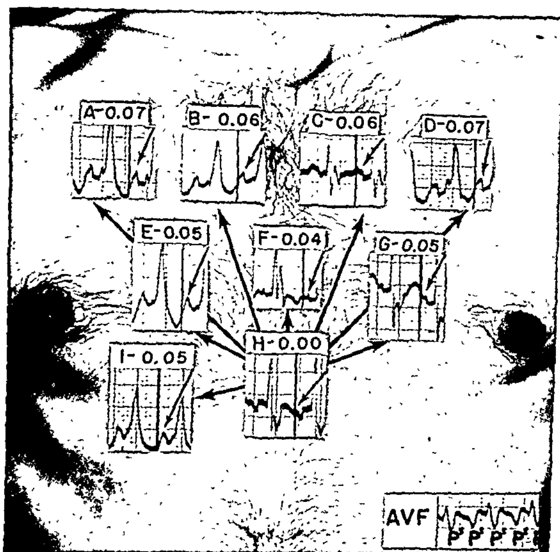


Figure 173 Chest leads from a patient exhibiting the common type of flutter with clear intrinsicoid deflections. The focus is directly beneath the lower end of the sternum, as evidenced by the pure negative auricular deflection recorded from this site (0.00). The vertical line in each cardiogram was obtained from a fixed reference point in simultaneous lead AVF (right lower insert) Note that the intrinsicoid deflections occur later and the deflections assume increasing degrees of positivity as the cardiograms are taken from areas more distal from the focus The patient also displays right bundle-branch block

ond (Figure 174A). As shown in Figures 173 and 174B, no such phenomenon occurred; on the contrary, the time values varied directly with the distance of the electrode from the ectopic focus. Thus only evidence inconsistent with a circus movement is found in the precordial electrocardiograms of the two instances of flutter in which auricular intrinsic deflections from precordial leads could be studied

COURSE OF THE EXCITATION WAVE OF PAROXYSMAL AURICULAR TACHYCARDIA IN MAN

Experimentally produced auricular tachycardia in animals (Chapter III) has been found to consist of rapidly recurring waves arising from an ectopic focus and spreading in all available directions simultaneously Identical observations on the nature of tachycardia were made in human subjects in whom the arrhythmia was produced experimentally during surgical procedures in the thorax (Chapter IV). As noted in Chapter III, workers are not yet in complete accord concerning the mechanism of clinical tachycardia; some believe a circus movement is involved, others do not In the present investigation the mechanism of tachycardia in man was studied by the same methods of esophageal lead electrocardiography applied to clinical flutter.

common type of flutter as shown in Figures 167 and 170 In both instances the auricular deflections clearly indicate that the impulse starts at an ectopic focus at the caudal end of the auricle and travels toward the cephalic end. In auricular paroxysmal tachycardia, as in flutter, the greater the distance between the esophageal electrode and the focus, the later is the time at which the intrinsicoid deflection from the electrode is recorded. Thus, in both arrhythmias the impulse starts at an ectopic focus and travels away from that focus in all available directions. In neither arrhythmia is there evidence of circus movement.

The electrocardiograms from esophageal leads in tachycardia in man are identical in form whether the arrhythmia occurs spontaneously or is experimentally produced (Chapter IV).

DISCUSSION

The foregoing studies of flutter and tachycardia in esophageal leads from humans, when correlated with our cinematographic and electrocardiographic observations of these arrhythmias in animals (Chapters V and VI), indicate that the waves of the two arrhythmias travel in the same manner in man as in the dog. The wave of flutter established at the caudal end of the auricles of dogs travels in a caudocephalic direction in the right and left auricles and the septum simultaneously. In man, despite complicated anatomic relationships, the flutter wave in esophageal lead electrocardiograms from various auricular levels exhibits a gradual, progressive change in configuration (similar to a simple muscle strip) as the recording electrode is moved beneath the auricles from one auricular extremity to the other. This is true whether the impulse arises from a focus at the cephalic end of the auricles and moves caudad, or whether it arises at the caudal end and moves cephalad The circus movement theory postulates that the course of the flutter wave in each auricle is different, that it travels upward in one auricle and downward in the other. Such a change in direction of the impulse would necessarily be reflected in an abrupt reversal in direction of the electrocardiographic deflections in esophageal leads from different auricular levels. No such change in direction of the impulse is recorded. Our analysis of esophageal lead tracings clearly demonstrates that each impulse originates at the focus and travels out over both auricles.

SUMMARY AND CONCLUSION

The excitation waves of auricular flutter and auricular paroxysmal tachycardia in man have been studied by means of esophageal and multiple precordial lead electrocardiography. The excitation waves of the two arrhythmias in man, as in the dog, are identical in that in each instance they arise from an ectopic focus and travel in all available directions simultaneously. No circus movement occurs.

Lewis calculated the course of the flutter wave in man from the momentary atrial electrical axes and believed he had demonstrated that the impulse traveled continuously in a circular manner around the auricles. Lewis was unaware of the fact that the fluter wave consists of a P′ wave of depolarization and a Ta wave of repolarization. When his data are reevaluated to include only the wave of excitation (P′), it is shown that the course of the flutter wave in his patient did not pursue a circus pathway.

Esophageal tracings were recorded from the levels of the caudal, the middle and the cephalic portions of the auricles in each of 10 instances of clinical flutter. The first group consisted of seven patients and represented the common type of flutter in which the P′ waves were inverted in limb leads 2, 3 and AVF, and were of low amplitude or isoelectric in lead 1. This group exhibited pure negative deflections in esophageal leads from the caudal level of the auricle, biphasic deflections from the mid-auricular level, and pure positive deflections from the cephalic level of the auricle. These observations indicate that the excitation wave of the common type of flutter arises at an ectopic focus in the caudal end of the au-

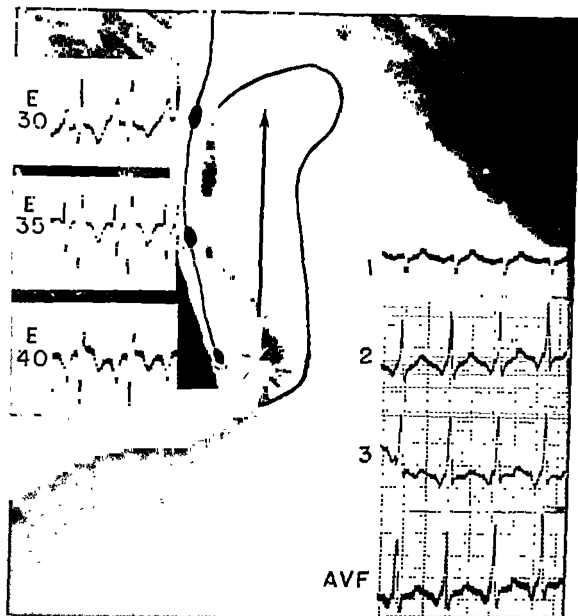


Figure 175 Three simultaneous esophageal electrocardiograms (E 30, E 35 and E 40) in a patient with auricular tachycardia. At E 40 a pure negative deflection is present At E 35 the negative deflection is preceded by a positive wave. At E 30 only a positive deflection is present. In this instance the impulse starts at the caudal end of the auricle and travels in a cephalic direction For reasons given in the text, existence of a pure positive deflection at E 30 precludes the possibility of a circus movement as the mechanism of paroxysmal auricular tachycardia. The P′ waves are inverted in the standard limb leads and lead AVF (below right) just as in auricular flutter from the caudal end of the auricles

The literature contains many examples of esophageal lead electrocardiograms of tachycardia in man[74, 86, 166, 263] Additional esophageal tracings of both spontaneous and experimentally produced tachycardia (Chapter IV) have been gathered from our own patients Examination of all these records consistently revealed that the auricular deflections of clinical tachycardia have the same configuration as those of flutter except for the usual absence of Ta waves. In both the spontaneous and the experimental varieties of human tachycardia the auricular deflection in esophageal leads is either a simple negative, a biphasic, or a simple upright deflection. No complicated multiphasic deflections (such as the series of the two positive-negative waves described on page 168), which would have to be present if a circus movement existed, were found. The record in Figure 175 is typical of that obtained from esophageal leads in human tachycardia and is analogous to the record from the corresponding study of the

CHAPTER IX

Further Clinical Observations On Auricular Flutter and Tachycardia

The exact criteria for a specific diagnosis of flutter, and less often of tachycardia, vary considerably in different clinics and in different textbooks. Indeed, one authority will label a record as flutter, while an equally competent cardiologist will interpret the same or an identical record as tachycardia. This chapter presents (1) a critical discussion of current criteria for the diagnosis of auricular tachycardia and flutter, and (2) a proposed classification of flutter and tachycardia designed to minimize present confusion.

PREVAILING CONCEPTS CONCERNING THE DIAGNOSIS OF FLUTTER

The following criteria have become more or less established in various clinics.

Auricular Rate: The statement has been made that flutter cannot exist unless the auricular rate is 250 or more beats per minute; if the rate is slower, the arrhythmia is diagnosed as tachycardia.

This arbitrary division based on rate is erroneous. We observed a patient with flutter in whom the auricular rate increased gradually from 200 to 300 beats per minute during the same paroxysm In four other instances similar variations in rate were noted during a single paroxysm The electrocardiograms recorded throughout a given paroxysm were virtually identical except for the changes in rate and the presence of Ta waves during the more rapid phases of activity. According to the aforementioned criterion, these patients would have two separate arrhythmias during the same paroxysm as a result of a simple change in rate. Tachycardia would be present while the auricular rate was 200 beats per minute and flutter while the rate was 300 beats per minute. Such examples demonstrate the worthlessness of a distinction based on rate alone (Figure 176).

Configuration of Auricular Deflection: Other cardiologists stress the shape of the auricular deflection in the diagnosis of the arrhythmia. If the record has an undulating, never-resting appearance, even if the rate is comparatively slow, the disturbance is considered to be flutter. On the other hand, if the P′ wave is followed by an isoelectric period and has little or no undulation, even if the rate is rapid, these cardiologists make a diagnosis of tachycardia.

In our experience this differentiation is meaningless. It is generally true that the faster the rate, the more conspicuous is the Ta wave and the more prominent the undulation. However, we have seen electrocardiograms in which the undulations are present in some leads and absent in other leads recorded during the same attack. Figure 177 shows the record from a patient with complete heart block and an auricular rhythm from an ectopic focus at a rate of 165 beats per minute. Undulations are present in lead V_1 but absent in lead 3. Obviously, the patient cannot have flutter (lead V_1) and tachycardia (lead 3) at the same rate during the same attack.

Figure 178 is the record from a patient with flutter at a rate of 214 auricular beats per minute. In lead 1 there are only P′ waves followed by isoelectric periods, whereas in simultaneously recorded lead 3 typical undulations are present.

ricles, travels in a caudocephalic direction and terminates at the cephalic end of the auricles.

A second group of two patients represented the uncommon type of flutter in which the P′ waves are upright in limb leads 1 and 2 and in lead AVF. In this group pure negative deflections were recorded in esophageal lead tracings from the cephalic level of the auricle and pure positive deflections were obtained from the caudal level. Thus, in the uncommon type of flutter the excitation wave originates at the cephalic end of the auricle, travels in a cephalocaudal direction and terminates at the caudal end.

The tenth instance of flutter exhibited at different times both the common and uncommon types of flutter. On one occasion inverted P′ waves were inscribed in limb leads 2, 3 and AVF; the auricular deflections from esophageal leads recorded on that occasion were identical with those observed in the seven instances of the common type of flutter, indicating that the wave arose at the caudal end of the auricle. On another occasion the P′ waves were upright in leads 1, 2, 3 and AVF; esophageal lead tracings obtained at that time were identical with those of the two instances of the uncommon type of flutter, revealing that the wave arose at the cephalic extremity.

The appearance of auricular deflections from esophageal leads in a hypothetical example of *circus movement* have been described. In our study and to our knowledge, auricular deflections from esophageal leads resembling those postulated have never been recorded. The *configurations of the deflections in esophageal lead* electrocardiograms of flutter obtained from our patients consistently indicate that the flutter excitation wave proceeds from the ectopic focus to the opposite end of the auricles and terminates; it does not return to the original focus.

The speed of the flutter excitation wave as it travels a fixed known distance has been roughly calculated. The rate of propagation of the excitation wave of flutter apparently is slower than that of the wave of normal sinus rhythm.

In two instances of the common type of flutter the course of the excitation wave, as determined by multiple precordial lead electrocardiography, has been traced over the chest. By timing the onset of the intrinsicoid auricular deflections it has been shown that the impulse was found to start at that 0.00 point and spread away from it, not in a narrow circular path, but in all available directions simultaneously.

Auricular tachycardia in man, either spontaneous or experimentally produced during thoracic surgical procedures, was analyzed by a method of electrocardiography similar to that used to investigate flutter. It was found that the *electrical events of tachycardia are similar to* those of experimental and clinical flutter, in each instance the impulse originates at an ectopic focus and travels concentrically from it. In neither arrhythmia is there a circus movement.

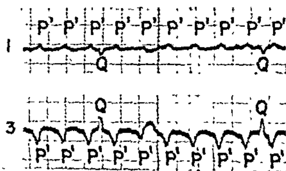


Figure 178 Electrocardiograms of simultaneously recorded leads 1 and 3 from a patient with auricular flutter at a rate of 214 beats per minute Ta waves are present in lead 3 but absent in lead 1

is present in lead 2 and 3 but not in lead 1. According to the sine wave criterion, this patient would have flutter in lead 2 and 3 but not in lead 1

Auriculo-Ventricular Block: Certain workers consider the presence of auriculo-ventricular block essential to the diagnosis of flutter. If block is present, the arrhythmia is commonly identified as flutter, if block is absent, the diagnosis is tachycardia

This appears to be an artificial distinction. The presence or absence of auriculo-ventricular block does not depend upon a difference in mechanism per se, but rather on the efficiency of the auriculo-ventricular node and conduction system. Auriculo-ventricular block may be physiological due to the high auricular rate as in flutter or due to a pathological interruption of the conduction system The latter has no relation to auricular rate. Several studies have been made of so-called tachycardia with block on the one hand, and 1·1 flutter on the other. We have shown in Chapters III and VII that most records interpreted as 1:1 flutter present no basis for this diagnosis. The undulations which appear between the QRS complexes are generally due to the ventricular T waves and the auricular deflections cannot be identified (Figure 180) It is these undulations which are the source of confusion.

The worthlessness of a distinction based on auriculo-ventricular block is best seen in the example of complete heart block where the pure auricular deflection is evident We have shown in these instances that the rate can fluctuate from slow to fast without any possible arbitrary distinction between tachycardia and flutter (Chapter VII).

PREVAILING CONCEPTS CONCERNING THE DIAGNOSIS OF TACHYCARDIA

The following are some of the commonly accepted criteria for the diagnosis of tachycardia.

Auricular Rate: The rate must be between 150 and 250 beats per minute.

The concept that tachycardia cannot exist at a rate below 150 also is incompatible with clinical experience. We have repeatedly seen short runs of tachycardia at auricular rates of approximately 90 beats per minute. Most such slow rate paroxysms last only a few beats and cause few or no symptoms (Figure 181). Theoretically, tachycardia can occur at a rate only a few beats per minute faster than normal sinus rhythm. This has been observed repeatedly, both experimentally and clinically. The slow rate tachycardias as a rule are diagnosed only by chance, by means of a routine electrocardio-

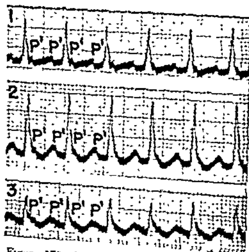


Figure 179 Leads 1, 2 and 3 from a patient with 2 1 auricular flutter An upright P' wave is present and there is a brief isoelectric interval in lead 1. A pure sine wave is seen in lead 2 and 3, the P' waves cannot be identified with certainty in these leads, and no isoelectric interval can be seen According to some electrocardiographers flutter can be diagnosed only if the sine wave is present This concept is erroneous, in the present instance, if the concept were valid, flutter would be present in leads 2 and 3 but not in lead 1

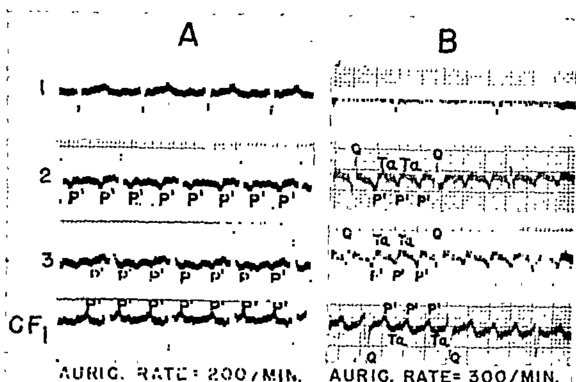


Figure 176 Auricular flutter of the common type with P' waves inverted in leads 2 and 3, and upright CF_1 In A the auricular rate is 200 beats per minute. The Ta waves are more distinct in B, when the auricular rate is 300 beats per minute

According to clinicians who base their diagnoses on the shape of the auricular deflection, the patient has tachycardia in lead 1 and flutter in lead 3. Many other examples of this situation are available.

In auricular arrhythmias, whether slow (generally diagnosed as tachycardia) or fast (generally diagnosed as flutter), Ta waves which cause the undulations may or may not be present. Thus, the concept that undulations are essential to the diagnosis of flutter and incompatible with a diagnosis of tachycardia is erroneous

Sine Wave: Some clinicians diagnose flutter only if the auricular deflection has the appearance of a pure sine wave.

If flutter is diagnosed only where the auricular deflection creates a pure sine wave, the great majority of these arrhythmias (approximately 95 per cent) will not be recognized, for such a wave is rare. Whether or not a sine wave is present depends largely on the rate. It occurs as a rule only with the rapid flutters or after administration of certain drugs such as quinidine; even under such circumstances the small isoelectric "shelf" may often be found. Obviously, the absence of a sine wave is irrelevant in the diagnosis of flutter. Figure 179 shows records from a patient with flutter, a sine wave

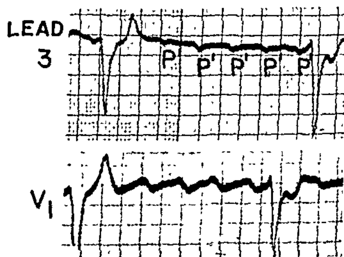


Figure 177. Successively recorded leads 3 and V_1 from a patient with coronary artery disease, posterior myocardial infarction, complete auriculo-ventricular block, and a regular ectopic auricular rhythm at rate of 165 beats per minute. The Ta waves are not present in lead 3 and only the inverted P' waves may be seen Ta waves are present in lead V_1 and give rise to marked undulations Thus in the same patient undulations may be present in one lead and absent in a successively recorded lead. The presence or absence of flutter undulations is determined solely by the presence or absence of Ta waves.

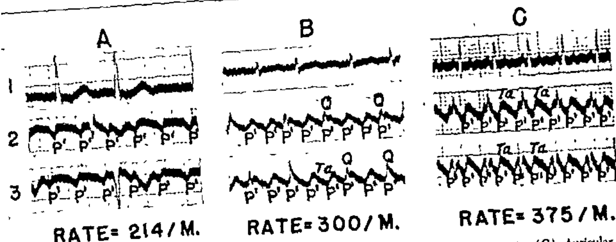


Figure 182 Examples of the common type of flutter in three patients, each with a different auricular rate.
(A) Auricular rate 214 beats per minute, *no* Ta waves are present (B) Auricular rate 300 beats per minute, small Ta waves are present. (C) Auricular rates 375 beats per minute; large Ta waves are present. *Note that as the rate becomes more rapid, the undulations are more apparent and the records are more characteristic of flutter.*

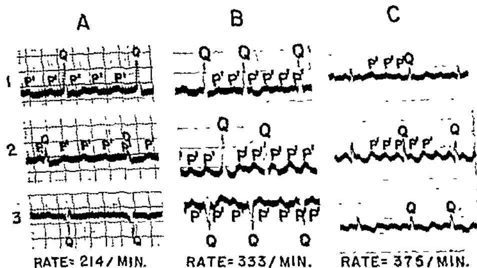


Figure 183 Electrocardiograms from three patients exemplifying the uncommon type of flutter with upright P' waves in leads I and 2. (A) Slow flutter with an auricular rate of 214 beats per minute. (B) More rapid flutter with an auricular rate of 333 beats per minute. (C) Fast flutter with an auricular rate of 375 beats per minute An isoelectric interval is present in A, characteristic undulations are distinct in B and C.

never made such sharp delineations as those which have in recent times become entrenched in many modern clinics

Clarification of the criteria for the diagnosis of flutter and tachycardia is obviously necessary. For this purpose, we have analyzed electrocardiographic records from 139 patients diagnosed as having auricular flutter and from 107 patients diagnosed as having auricular tachycardia by cardiologists at the Los Angeles County General Hospital and other institutions.

ANALYSIS OF ELECTROCARDIOGRAMS OF FLUTTER

Our study of records from 139 patients indicates that flutters may be classified into four more or less distinct types based upon the configuration of the auricular deflections. It is of

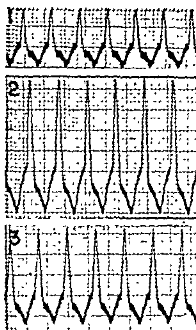


Figure 180. Leads 1, 2 and 3 from a patient with a rapid ectopic rhythm. The undulations seen between the QRS complexes are not identifiable, they are probably pure ventricular T waves. *Records of this type* are often diagnosed as 1 1 flutter There is no sound basis upon which to diagnose this electrocardiogram as flutter, the nature of the disturbance is unknown from the available data. A diagnosis might be obtained by the use of esophageal or special chest leads.

gram. They cannot be diagnosed clinically.

Configuration of P' Wave: The P' wave must have an abnormal configuration.

This is not an essential criterion. In most instances the P' wave cannot be seen. If seen, it usually has an abnormal configuration. If the ectopic focus is at or near the sinus node, however, the P' wave may have a normal or almost normal configuration (Chapter VII).

Isoelectric Interval: An isoelectric interval must occur after the P' wave, and undulation must not be present.

This is not necessarily true; as pointed out above, an isoelectric interval may be present in some leads but absent in others during the same bout of the arrhythmia (Figures 177 and 178).

Auriculo-Ventricular Block: Auriculo-ventricular block does not exist in tachycardia when the auriclo-ventricular conducting system is normal.

This criterion has already been discussed in relation to flutter. It was shown that the ventricular response depends on the efficiency of the conducting system.

It would seem that many of the present criteria for the diagnosis of flutter and tachycardia are contradictory and practically meaningless. The consequent confusion regarding these two disturbances exists even in some of the leading clinics and among outstanding cardiologists.

Our studies of the mechanism of flutter and tachycardia, and a review of the historical evolution of knowledge on these subjects, reveals that the physiologic and historic bases for present concepts are obscure. Early authorities

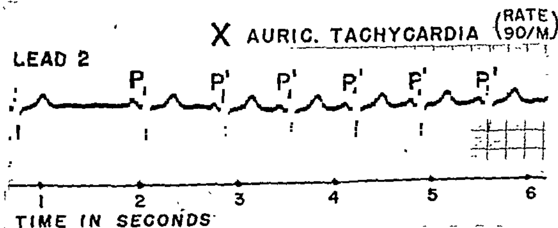


Figure 181. Lead 2 from a 75 year old female who complained only of palpitation The electrocardiogram showed short runs of auricular paroxysmal tachycardia at a slow rate of 90 beats per minute. The attacks generally lasted only a *few beats.*

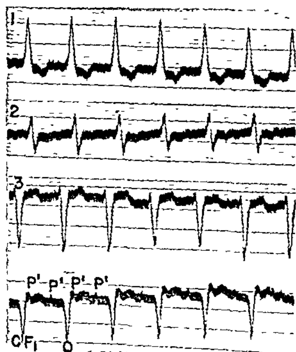


Figure 186. Auricular flutter with 2:1 block No auricular deflections are visible in standard leads 1, 2 and 3 This type of record is often diagnosed as nodal tachycardia In CF_1, however, it can be seen that the arrhythmia is 2 1 auricular flutter.

Flutter with the Focus at the Cephalic End of the Auricles at or Near the Sinus Node (Type II): In 21 of the 139 instances of flutter (15.1 per cent), the P′ waves were upright in leads 1 and 2; usually they were upright in lead 3 The P′ wave was similar to or identical with the normal P wave (Figure 183) This electrocardiographic picture represents a disturbance starting near the sinus node and traveling in a cephalocaudal direction (Chapters VII *and* VIII) For descriptive purposes this might be called "the cephalic type" of flutter.

Sine Wave Group (Type III): In six of the 139 instances (4.3 per cent), we could not identify with certainly the P′ and Ta waves in the standard limb leads, although the records were typical of flutter *These records* display continuous undulations, usually a sine wave, and no isoelectric "shelf" (Figure 184). In some instances this pattern may have been due *in* part to the effect of quinidine in accentuating the undulations The finding of an intrinsic deflection by unipolar chest or esophageal electrocardiography should delineate the P′ wave of this type of flutter.

Mimic Group (Type IV): In 16 of the 139 instances (11.5 per cent), the auricular activity reflected in the limb leads suggested erroneous diagnoses. From standard limb leads, the diagnosis of auricular fibrillation (Figure 185), nodal tachycardia (Figure 186), and auricular tachycardia (Figure 187), could be made. The diagnosis of flutter was made from chest leads. In several of these instances, the ventricular deflections obscured some of the auricular activity. Flutters of the type simulating tachycardia usually may be diagnosed by increasing the auriculo-ventricular block by carotid sinus pressure (Figure 188). In many of these instances

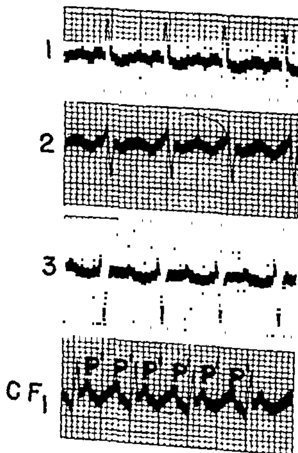


Figure 187 Auricular flutter simulating auricular paroxysmal tachycardia Only one P′ wave is visible for each ventricular complex in leads 1, 2 and 3. The true diagnosis is revealed in CF_1

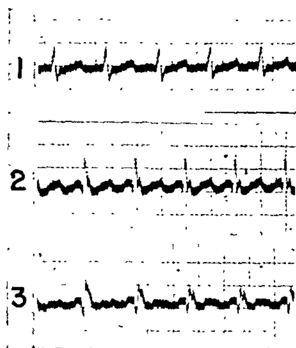
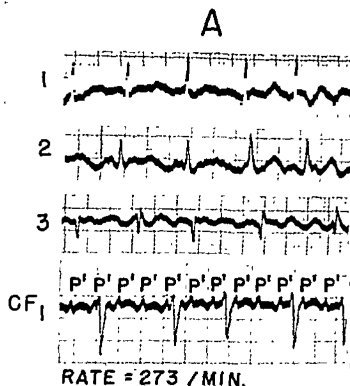


Figure 184 An example of the sine type of auricular flutter. The flutter deflection consists of a sine wave without an isoelectric interval The P′ and Ta waves cannot be identified with certainty. In this type of flutter the site of the ectopic focus cannot be ascertained from the standard limb leads, esophageal leads should be employed for this purpose.

interest that the P′ wave could be identified in standard limb leads in almost 90 per cent of these instances of flutter.

Flutter with the Focus at the Caudal End of the Auricles (Type I): This is the most common type of flutter found in 96 of the 139 instances of flutter (69.1 per cent), the P′ waves were deeply inverted in leads 2 and 3; large Ta waves were generally present and always oppositely directed (Figure 182). In an occasional patient in whom augmented unipolar limb leads were recorded, the P′ wave was inverted in lead AVF. We have shown in previous chapters (VII, VIII) that such disturbances start at the caudal end of the auricles; this is the common type of flutter with which every clinician is familiar. The P′ waves are usually identified with ease; if esophageal leads are recorded, the position of the focus and the direction in which the wave travels may be accurately predicted. Figure 182 shows several examples of this type of flutter at varying rates. For descriptive purposes this type of flutter might be called "the caudal type."



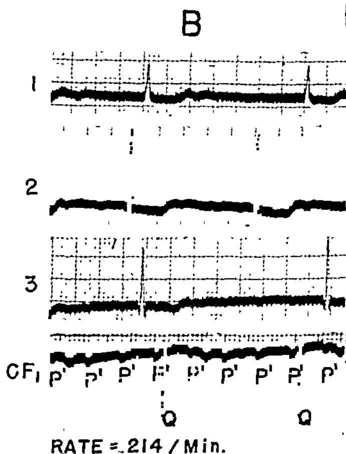


Figure 185 (A) and (B) Examples of auricular flutter mimicking fibrillation in standard leads 1, 2 and 3 No flutter wave can be seen in these leads. The true nature of the arrhythmia is revealed in CF_1

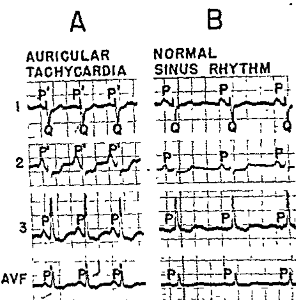


Figure 190 (A) Auricular paroxysmal tachycardia with focus of origin in the cephalic portion of the auricles near the sinus node. The P' waves are upright in leads 1, 2 and 3, and AVF (See esophageal leads of this patient in Figure 88)

(B) After restoration of normal sinus rhythm. The P waves are upright in leads 1, 2 and AVF, but of different configuration than the P' waves of the tachycardia The P wave is now inverted in lead 3

right in leads 1 and 2 and either upright or inverted in lead 3 (Figures 43 and 190). In a few patients in whom augmented unipolar limb leads were recorded, the P' wave in lead AVF was upright The P' waves in this type of tachycardia are similar to those of the cephalic type of flutter.

Tachycardia in which the Site of Origin is Indeterminable (Type III): In 66 of the 107 instances (61 8 per cent), little or no definite evidence of auricular activity was clearly discernible. Probably many or most of these instances are actually the cephalic type with upright P' waves inscribed in the preceding ventricular T waves and often with prolongation of the P'-R intervals (Figure 191). It is unlikely that these instances represent tachycardia of the caudal type since the sharply inverted P' wave is characteristic of the latter group and is easily visible as a sharp downward notching in or immediately before the T wave; this we have actually seen. As shown in Figure 52, if the P' wave were upright it would have merged with the ventricular T wave and an anatomic diagnosis would have been impossible.

The P' wave in the cephalic type of tachycardia can best be seen when the rate is relatively slow and the P'R interval relatively short. It then appears as an upright wave following or merging with the ventricular T wave. As the rate of the tachycardia becomes more rapid, the P'-R interval usually becomes progressively longer until block occurs. The P' wave in rapid tachycardia therefore may be buried in the preceding ventricular T wave In two such instances the P' wave could not be identified during the paroxysm but was seen during the onset of tachycardia; in both, the wave was upright (Figure 192). The caudal type of tachycardias can frequently be identified, even when the rate is rapid, as the P' wave is a sharp, inverted spike which often appears just before or after the ventricular QRS complex (Figure 193).

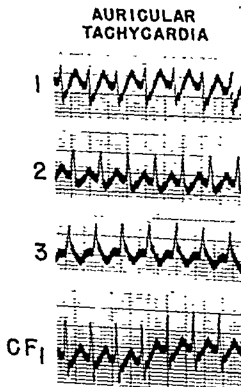


Figure 191 Auricular ... Th... it i... lar ... of this type

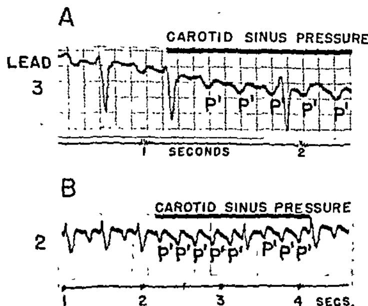


Figure 188. Auricular flutter simulating auricular paroxysmal tachycardia.

(A) The first two ventricular beats are preceded by P' waves, *this pattern could be diagnosed as auricular paroxysmal tachycardia.* Following carotid sinus pressure auriculo-ventricular block increases and the characteristic appearance of flutter becomes apparent.

(B) As in A, the first three beats resemble tachycardia. After carotid sinus pressure the rapid auricular activity is apparent.

In each of these records, before carotid sinus pressure was applied, *the alternate P' waves are buried in QRS complexes*

the ectopic focus probably was in the center of the auricles. Under such circumstances we have found small or isoelectric P' waves (Chapter IV).

ANALYSIS OF ELECTROCARDIOGRAMS OF TACHYCARDIA

Three general types of tachycardia based on the configuration of the auricular deflection were distinguished among the records from 107 patients included in the study. *In 65 per cent of the records studied, the P' wave could not be identified in the usual limb leads.*

Tachycardia Arising at the Caudal End of the Auricles (Type I): As has been shown, when the focus is at the caudal end of the auricle, the P' waves are inverted in leads 2 and 3 *(Figure 189). Ten of the 107 instances of* tachycardia (9.3 per cent) were of this type. The P' waves resembled the P' waves of flutter from the same site *(Type I flutter). This might* be called "the caudal" type.

Tachycardia *Arising* at the Cephalic End or at or Near the Sinus Node (Type II): *This* type of tachycardia was observed in 31 of the 107 patients (28 9 per cent). The P' waves were up-

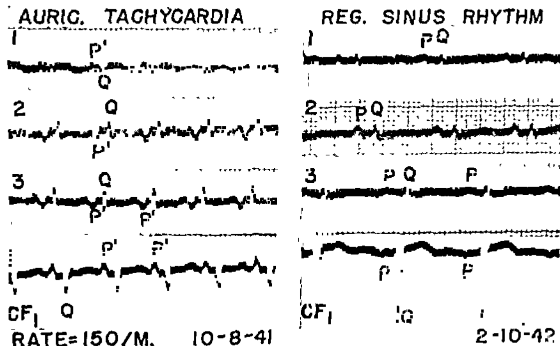


Figure 189 (A) Auricular paroxysmal tachycardia arising at the caudal *portion of the auricle. The P' waves are inverted in leads* 2 and 3, and upright in CF_1. *(B)* After restoration of normal sinus rhythm, the P' waves are upright in leads 2 and 3 and inverted in CF_1

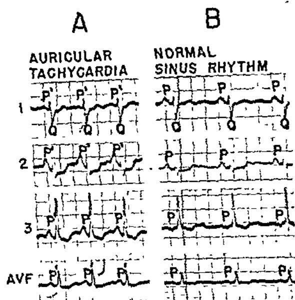


Figure 190 (A) Auricular paroxysmal tachycardia with focus of origin in the cephalic portion of the auricles near the sinus node The P′ waves are upright in leads 1, 2 and 3, and AVF (See esophageal leads of this patient in Figure 88)

(B) After restoration of normal sinus rhythm. The P waves are upright in leads 1, 2 and AVF, but of different configuration than the P′ waves of the tachycardia. The P wave is now inverted in lead 3.

right in leads 1 and 2 and either upright or inverted in lead 3 (Figures 43 and 190) In a few patients in whom augmented unipolar limb leads were recorded, the P′ wave in lead AVF was upright. The P′ waves in this type of tachycardia are similar to those of the cephalic type of flutter.

Tachycardia in which the Site of Origin is Indeterminable (Type III): In 66 of the 107 instances (61.8 per cent), little or no definite evidence of auricular activity was clearly discernible. Probably many or most of these instances are actually the cephalic type with upright P′ waves inscribed in the preceding ventricular T waves and often with prolongation of the P′-R intervals (Figure 191). It is unlikely that these instances represent tachycardia of the caudal type since the sharply inverted P′ wave is characteristic of the latter group and is easily visible as a sharp downward notching in or immediately before the T wave; this we have actually seen As shown in Figure 52, if the P′ wave were upright it would have merged with the ventricular T wave and an anatomic diagnosis would have been impossible.

The P′ wave in the cephalic type of tachycardia can best be seen when the rate is relatively slow and the P′R interval relatively short. It then appears as an upright wave following or merging with the ventricular T wave. As the rate of the tachycardia becomes more rapid, the P′-R interval usually becomes progressively longer until block occurs The P′ wave in rapid tachycardia therefore may be buried in the preceding ventricular T wave. In two such instances the P′ wave could not be identified during the paroxysm but was seen during the onset of tachycardia; in both, the wave was upright (Figure 192). The caudal type of tachycardias can frequently be identified, even when the rate is rapid, as the P′ wave is a sharp, inverted spike which often appears just before or after the ventricular QRS complex (Figure 193).

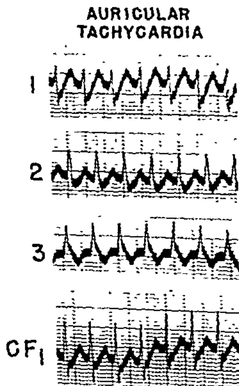


Figure 191 Auric...

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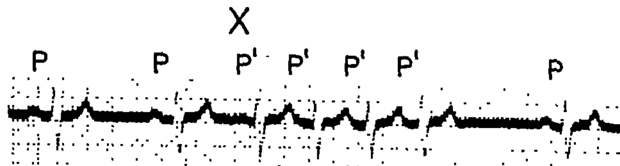


Figure 192 Lead 2 Short runs of auricular paroxysmal tachycardia. The P' waves cannot be seen during the run but can be identified as the first beat (X) of the paroxysm is recorded. The P' wave here is upright and the impulse arises in the cephalic end of the auricles.

Some of the tachycardias classified as Type III probably arise from a focus midway between the caudal and cephalic ends of the auricle. As shown in Chapter IV, esophageal lead records of experimental tachycardia and premature systoles in man produced from this site have small or isoelectric P' waves; consequently, P' waves would only rarely be visible in the limb leads. Finally, it is possible that some tachycardias assigned to the indeterminate type, while they actually represent the caudal or cephalic type, cannot be properly identified as the P' wave is obscured by an abnormal rotation of the heart (Chapter VII).

Thus, of the tachycardias designated as the indeterminate type, probably the majority are in fact the cephalic type and a minority are the caudal type. The percentage of tachycardias actually of the cephalic type (tachycardias from near the cephalic end of the auricle), undoubtedly is underestimated.

SITE OF THE ECTOPIC FOCUS

As suggested by our statistical analysis, in most instances a fundamental anatomic difference exists between tachycardia and flutter: this difference consists in the location of the ectopic foci. The majority of flutters originate at the caudal end of the auricles, only a relatively small percentage of tachycardias start in this region. Relatively few flutters arise in the cephalic region; of those tachycardias in which the site of origin is identifiable, the majority usually start in this area If, as seems likely, the most common sites of origin of the two arrhythmias are widely separated, this anatomic predilection may account for some of their clinical dissimilarities. The auricular rate is usually faster in flutter than in tachycardia. Flutter generally persists for a longer period and is more difficult to terminate than is tachycardia. Usually physiologic auriculo-ventricular block is the rule in flutter but does not occur in tachycardia If block is present in auricular paroxysmal tachycardia it is pathologic. From this a new and more accurate definition of flutter may be drawn. *Flutter is a fast auricular tachycardia usually starting at the caudal end of the auricles with a physiologic auriculo-ventricular block.*

EFFECT OF VAGAL STIMULATION

The fact that vagal stimulation has different effects on auricular paroxysmal tachycardia from those on auricular flutter has been advanced as proof that the mechanisms of these two arrhythmias are different. The following considerations suggest that the response to vagal stimulation may vary with the rate rather than the mechanism of the arrhythmia.

The statement that carotid sinus stimulation is effective in auricular tachycardia and not in auricular flutter is not entirely true. Clinically and experimentally, this procedure generally is ineffective in very rapid auricular paroxysmal tachycardia as well as in auricular flutter. We have repeatedly observed, as did Lewis, that vagal stimulation frequently terminates slow auricular paroxysmal tachycardia but rarely stops rapid auricular tachycardia. It is well known that the supra-ventricular tachy-

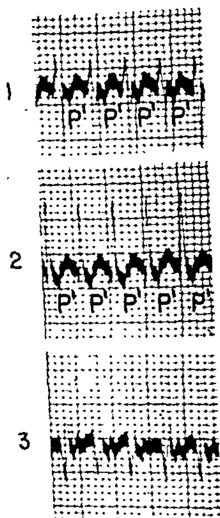


Figure 193 Auricular paroxysmal tachycardia with inverted P' waves, easily identified in spite of the rapid rate (EkG enlarged two times) If the P' waves were upright at this rate, the arrhythmia probably could not be identified with certainty

cardias such as occur in infants are resistant to carotid sinus massage Thus, carotid sinus stimulation tends to terminate those arrhythmias in which the ectopic focus is discharging slowly (slow auricular paroxysmal tachycardia) and fails to affect those in which the ectopic focus is discharging rapidly (rapid tachycardia or flutter)

The importance of auricular rate in determining certain responses to vagal stimulation was shown many years ago by Lewis, who demonstrated that this procedure results in fibrillation only if it is applied while the auricles are beating at or above a critical rate, in most animals 400 per minute. From this experiment he concluded that "there is a very clear relation to the preliminary rate of beating."[371] Lewis also observed that vagal stimulation caused asystole only if the auricles were beating at a relatively slow rate, although he failed to emphasize the clinical significance of this finding We have confirmed Lewis' observations by administering acetylcholine to the experimental animal while the auricles were stimulated by electrical shocks at various rates. Asystole followed administration of acetylcholine when the auricles were being driven at the rate of a slow tachycardia, the agent precipitated fibrillation when the rate of stimulation was above 400 per minute (Figure 194). The same observation was made with aconitine induced arrhythmias (Figure 195). As Lewis and Masters noted, auriculo-ventricular conduction is related to auricular rate and under the influence of vagal stimulation, auriculo-ventricular block tends to develop at lower auricular rates than under normal circumstances.[377] Thus it is apparent that the response of an auricular arrhythmia to vagal stimulation cannot always be predicted but whether asystole or increase of auricular rate occurs is intimately dependent upon the auricular rate prevailing when the vagus is stimulated, while auriculo-ventricular block is most likely to be produced at rapid auricular rates. These experimental observations are a counterpart of the clinical observation that vagal stimulation has a different effect in slow tachycardia than in rapid tachycardia or flutter.

The fact that disturbances with identical mechanisms may respond differently to carotid sinus stimulation is apparent from a consideration of the effects of this procedure on normal sinus rhythm. In one individual auricular asystole may result while in another auriculo-ventricular block occurs In the first instance the response to vagal stimulation is similar to that generally occurring in slow tachycardia while in the second instance the response resembles that of flutter. However, the mechan-

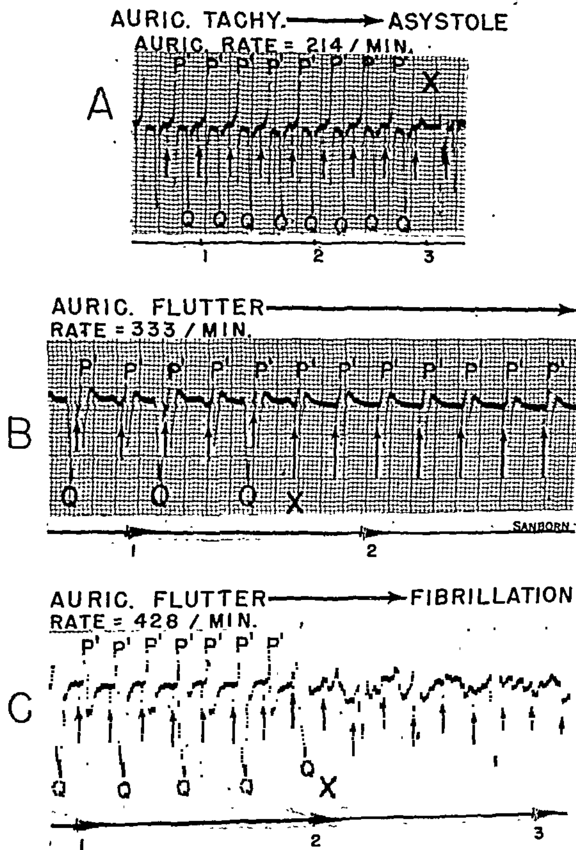


Figure 194. A. Auricular tachycardia produced by electrical stimulation at a rate of 214 per minute. Acetylcholine was administered intravenously while the auricle was being stimulated. After the injection of acetylcholine, asystole (X) occurred. (Recorded at 25 mm per second.)

B. Auricular flutter produced by stimulating the auricle electrically at a rate of 333. While the auricle was being stimulated, acetylcholine was administered. Note the auricles continued to respond but that ventricular asystole occurred. (Recorded at 50 mm per second.)

C. Auricular flutter produced by stimulating the auricle at a rate of 428 per minute. Again while the auricle was being stimulated acetylcholine was administered. The rhythm now converted to auricular fibrillation. (Recorded at 50 mm per second.)

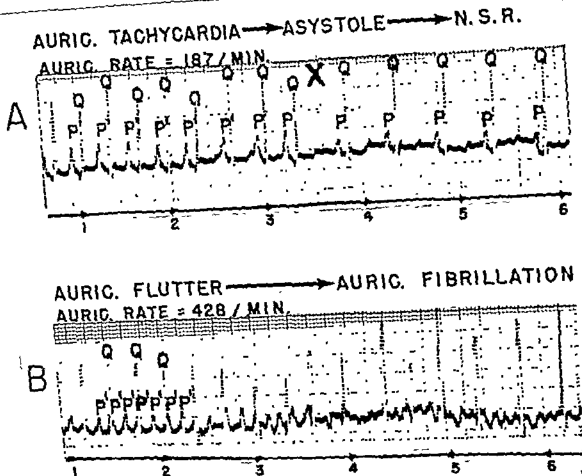


Figure 195 A Auricular tachycardia (rate 187 per minute) produced by aconitine Acetylcholine was injected intravenously. This caused auricular asystole (X) Regular sinus rhythm then ensued.

B Auricular flutter (rate 428 per minute) produced by aconitine. After acetylcholine was administered the rhythm converted to fibrillation

ism of normal *sinus rhythm obviously is the* same in the two individuals; only the response of the heart, and especially of the conduction system, has varied. By analogy, the difference in response to vagal stimulation does not constitute proof of a fundamental difference between auricular paroxysmal tachycardia and auricular flutter.

In most individuals with auricular paroxysmal tachycardia there appears to be a firm binding of unknown nature between the auricles and ventricles, as shown by the absence of auriculo-ventricular block, in these instances carotid sinus stimulation does not cause the development of auriculo-ventricular block The presence of this firm binding is evidenced by the fact that in a given patient carotid sinus stimulation may cause prolongation of the P-R interval after termination of a paroxysm of tachycardia but not during the bout. On the other hand, in auricular flutter the firm binding is already *broken; here*, increase in vagal tone causes an increase in the existing block. An explanation of these differences must await elucidation of the auriculo-ventricular conduction system. That a more complex process is involved than simple conduction between auricle and ventricle has been shown by our observations of aberration and auriculo-ventricular conduction (Chapter XV). Until further knowledge of auriculo-ventricular conduction is obtained, the concept of the unitary nature of clinical auricular tachycardia and auricular flutter must remain a theory.

DISCUSSION

Although the preceding observations to some extent clarify the relationship between clinical tachycardia and flutter, they also emphasize that many records present characteristics of both arrhythmias. In every electrocardiographic department, tracings are frequently obtained which are a source of argument as to whether the record represents tachycardia or flutter. Such arguments are meaningless, and dogmatic opinions are unwarranted. It is suggested that those borderline cases which present characteristics of both arrhythmias, rather than being designated by name, might more profitably be described in terms of the disturbed function. In such instances the following type of description should prove useful: (1) the auricular rate should be stated; (2) the configuration of the P′ wave should be described; (3) if possible, position of the ectopic focus should be indicated; (4) the description of a Ta wave should be given; (5) auriculo-ventricular conduction (presence or absence of block) should be mentioned; and (6) presence or absence of ventricular aberration should be specified.

In all instances, whether debatable or subject to definite diagnosis, the physiologic aspects rather than the name of the disturbance deserve primary emphasis. Cardiologists 30 years ago made the simple diagnosis of "coronary occlusion." Today, a more descriptive terminology is used, including such data as whether or not tissue destruction is present, the size of the infarction, the part of the heart that is involved, whether the electrocardiographic changes resulted from coronary occlusion or insufficiency. The addition of these details, learned through considerable experience and investigation during the past several decades, has been of tremendous aid to both patient and physician.

Likewise, a description of the physiologic characteristics of the disturbance in each particular auricular arrhythmia, although lengthy, has certain virtues which are of more than theoretical importance. When sufficient data have been obtained, correlation of variations in electrocardiographic configurations with variations in the clinical course of the arrhythmias may reveal the prognostic and therapeutic implications of each electrocardiographic characteristic. In addition, the descriptive diagnosis provides a picture of what is actually happening in the auricles rather than a mere term which often causes meaningless controversies.

The term "flutter," signifying rapid and regular contractions, was first used by MacWilliam in reporting experiments on animals and later by Jolly and Ritchie to describe a clinical entity. This term is equally descriptive of the auricular activity in a rapid tachycardia, for the auricles during flutter and tachycardia are essentially identical electrocardiographically and cinematographically. Clinical usage tends to associate rapid beating of the ventricles with the term auricular paroxysmal tachycardia. In complete auriculo-ventricular block, however, the ventricles do not beat rapidly, even in the presence of auricular tachycardia. Contrariwise, in flutter with little or no block, the ventricles may beat rapidly. Thus, in the light of present knowledge of the arrhythmias, it would seem that the terms of flutter and tachycardia as originally defined are no longer appropriate.

We recommend that the term, *auricular tachycardia,* be retained; however, it should be redefined to include all arrhythmias now diagnosed as auricular tachycardia, auricular flutter, or borderline cases. It is proposed that auricular tachycardia be defined as a regular auricular rhythm occurring without *physiologic* auriculo-ventricular block and arising from an ectopic auricular focus whose rate of discharge is faster than the sinus rate. If the auricular rate is very rapid with resulting *physiologic* auriculo-ventricular block, such a condition might be designated, in deference to traditional usage, auricular flutter with the understanding that the basic mechanism is auricular tachycardia. Since nodal tachycardia can never be diagnosed with surety, considerable hesitation should be employed before utilizing this term.

In order to emphasize the differences between various types of arrhythmias while avoiding arbitrary distinctions based on questionable criteria, the general diagnostic term "auricular tachycardia" should be supplemented by a description of the physiologic disturbance. As a hypothetical example, the descriptive diagnosis of a particular instance of auricular tachycardia might be as follows: Regular auricular rhythm from an ectopic focus; rate 300 beats per minute; P′ waves inverted in leads 2 and 3; focus at the caudal end of the auricle; upright Ta wave in lead 1; followed by a short isoelectric shelf, varying 2:1 auriculo-ventricular block, marked varying ventricular aberration Wherever possible, the descriptive diagnosis should include a comparison of the electrocardiogram during the arrhythmias with that during normal sinus rhythm in the same patient. In the next chapter, examples of this type of description are given.

SUMMARY AND CONCLUSION

Many erroneous and conflicting opinions concerning the exact criteria necessary for differentiating auricular flutter from tachycardia are found among electrocardiographers and in various clinics and textbooks. These criteria mainly concern the auricular rate, the configuration of the P′ wave, and the presence or absence of auriculo-ventricular block. What may be called flutter in one institution is dogmatically asserted to be tachycardia in another. An analysis of the reasoning behind such viewpoints reveals their lack of substantial physiologic bases.

In order to clarify the relationship between these two arrhythmias, records from 139 cases of flutter and 107 cases of tachycardia were analyzed.

Four types of auricular deflections are found in electrocardiograms of flutter In the great majority of instances the P′ waves are inverted in leads 2, 3 and AVF, indicating that the ectopic focus is at the caudal end of the auricles and the impulse travels in a caudocephalic direction In a minority of instances the focus is at the cephalic end and the impulse travels in a cephalocaudal direction; here the P′ waves are upright in leads 1 and 2. Occasionally, electrocardiograms of auricular flutter are of the "sine-wave" type in which the P′ wave cannot be identified with certainty unless chest or esophageal leads are recorded. In the few remaining instances the diagnosis of flutter is divulged only in chest or esophageal leads; standard leads 1, 2 and 3 may mimic auricular fibrillation, nodal tachycardia, or auricular tachycardia.

In two-thirds of the instances of tachycardia the P′ waves cannot be identified in the limb leads. This is accounted for by the fact that the P-R interval becomes greatly prolonged when the auricular rate increases. Of these tachycardias in which the P′ wave may be identified, the majority start in the cephalic portion and display upright P′ waves in leads 1, 2 and AVF; a minority start at the caudal end and show inverted P′ waves in leads 2, 3 and AVF. Probably most tachycardias in which the P′ wave is unidentifiable originate at the cephalic end.

These data indicate an anatomic difference between tachycardia and flutter: in most instances of flutter the ectopic focus is at the caudal end of the auricles, whereas in tachycardia the site of predilection appears to be cephalic. This difference may explain certain clinical and therapeutic dissimilarities of the two disturbances. Differences in the response to vagal stimulation of slow tachycardia and flutter are related to a difference in rate rather than in mechanism of the disturbance.

The designation of an arrhythmia as "tachycardia" or "flutter" sometimes leads to confusion and fails to indicate what is actually happening in the auricle It is proposed that a physiologic description be substituted for the present practice of designating the disturbance by name alone The definition of "auricular tachycardia" might be altered to include all disturbances now diagnosed as flutter or tachycardia. To supplement this diagnostic term, the auricular rate should then be mentioned, the configuration of the P′ wave should be described; if possible, the site of the focus should be indicated; the Ta wave, if present, should be described, the pres-

ence or absence of auriculo-ventricular block should be noted; finally, the description should specify whether or not ventricular aberration is present. Such descriptive diagnoses not only have the advantage of avoiding confusion, but also provide information which aids in the understanding of the disturbance.

CHAPTER X

Examples of Physiologic Diagnosis of Auricular Tachycardia and Flutter

In the preceding chapter, attention was called to the arbitrary character of the nomenclature currently used in diagnosis of the auricular arrhythmias. The present chapter provides further evidence that the artificial criteria of present-day diagnosis of necessity result in ambiguity, *confusion* and *diversity* of diagnosis. In order to overcome these difficulties, we have designed a new method of electrocardiographic diagnosis The following type of description is suggested (1) The rate of discharge from the ectopic auricular focus should be stated, (2) the configuration of the P' wave should be described, (3) the description of a Ta wave should be included, (4) if possible, position of the ectopic focus should be indicated, (5) presence or absence of auriculo-ventricular block should be mentioned, (6) presence or absence of ventricular aberration should be specified; and (7) the diagnosis should conclude with an interpretation of the tracing summarizing the preceding six items. After the reader has become acquainted with the descriptive principles he can shorten this procedure of analysis by simply stating the summary interpretation

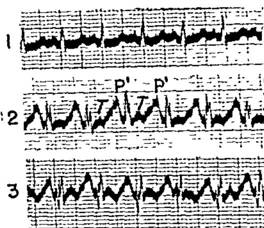
In order to evaluate this method of descriptive diagnosis and to compare it with present-day standard methods, we have applied both systems to a series of 32 selected electrocardiograms of various auricular arrhythmias ranging from simple to complicated types. Standard interpretations of this series of tracings were made by five leading and extremely able electrocardiographers in Los Angeles. It is important to point out that these five excellent electrocardiographers were trained in different centers throughout the United States.

It is astonishing that of the 32 tracings submitted to the five electrocardiographers and diagnosed by their standards, agreement was reached *in only 12 of the records*. In the remaining 21 records there was disagreement, in some instances of extreme degree, concerning the diagnosis. From a consideration of the physiologic aspects discussed in the preceding chapter, such differences are to be expected until the exact physiologic disturbance in each arrhythmia is defined. This chapter constitutes a practical demonstration of the defects of present nomenclature and of the advantages of describing an arrhythmia in terms of the disturbed physiologic function.

The 32 electrocardiograms illustrated in this chapter are classified into groups according to whether the ectopic focus of the arrhythmia is located at a high (Cases 1 to 10); low (Cases 10 to 24), middle or undeterminable auricular site (Cases 24 to 32); within each group the tracings are arranged in order of increasing auricular rate. The legends under each electrocardiogram include (1) a full description of the tracings; (2) our interpretations printed in bold type; (3) comments if indicated, and (4) the standard electrocardiographic diagnoses reported by the five electrocardiographers.

Standard interpretations of 24 of the electrocardiograms as reported by five experienced electrocardiographers appear in chart form in Figures 228 and 229. The charts are arranged in order of increasing rate of discharge from the

ence or absence of auriculo-ventricular block should be noted; finally, the description should specify whether or not ventricular aberration is present. Such descriptive diagnoses not only have the advantage of avoiding confusion, but also provide information which aids in the understanding of the disturbance.



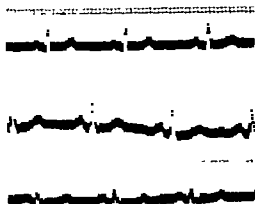


Figure 197 (Case No 2) Ectopic auricular tachycardia. Auricular rate 158 P' wave upright in lead 1, superimposed on T waves in leads 2 and 3 Ta waves absent or masked in leads 1, 2 and 3 Possible high auricular focus P'-R interval .12. Ventricular rate 158 No auriculo-ventricular block

INTERPRETATION: (A) AURICULAR TACHYCARDIA FROM HIGH AURICULAR FOCUS, AURICULAR RATE 158 (B) REGULAR SINUS RHYTHM.

Comment: P' waves superimposed on the T wave causes the appearance of a greatly prolonged Q-T interval

(5) Auricular tachycardia.

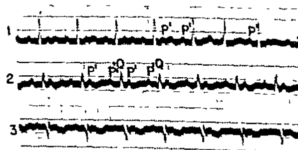


Figure 198 (Case No. 3) Ectopic auricular tachycardia. Auricular rate 187 P' wave upright in leads 1 and 2, low in 3 Ta waves absent High auricular ectopic focus Varying ventricular rate Regular 2 1 auriculo-ventricular block in leads 2 and 3, irregular lead 1. Slight ventricular aberration

INTERPRETATION. AURICULAR TACHYCARDIA WITH VARYING AURICULO-VENTRICULAR BLOCK, HIGH AURICULAR FOCUS, AURICULAR RATE 187.

Comment· This tracing is interesting because of the irregular block in lead 1 and the regular block in leads 2 and 3 It is tracings of this type that are presently debated whether to call flutter or tachycardia.

Electrocardiographers' Interpretation·

(1) Auricular tachycardia with 2 1 block.
(2) Auricular flutter with 2.1 block.
(3) Auricular tachycardia with 2 1 block.
(4) Auricular flutter with 3 1 block
(5) Auricular tachycardia with 2 1 block.

auricular ectopic focus. Electrocardiograms of arrhythmias from a high auricular focus are represented in Figure 228 and those from a low auricular focus in Figure 229. It is apparent that no sharp line of demarcation exists between tachycardia and flutter.

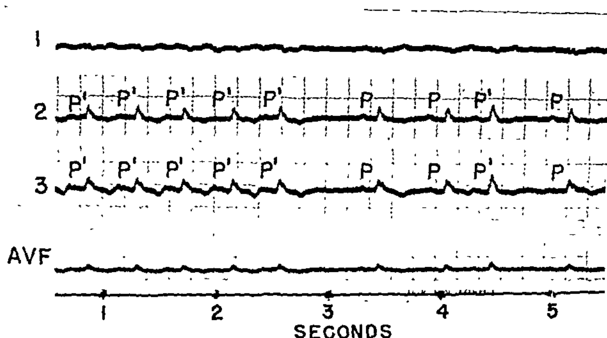


Figure 196 (Case No. 1) Ectopic auricular tachycardia with transition to regular sinus rhythm and auricular premature systole. Auricular rate 136. P' wave upright in leads 2 and 3, masked in lead 1. Ta wave masked in leads 1, 2 and 3 *High auricular ectopic focus*

INTERPRETATION: AURICULAR TACHYCARDIA FROM ECTOPIC FOCUS IN UPPER PART OF AURICLE, AURICULAR RATE 136, TRANSITION TO NORMAL SINUS RHYTHM AFTER 5TH BEAT WITH A PREMATURE SYSTOLE FROM SAME ECTOPIC FOCUS.

Electrocardiographers' Interpretation

(1) Auricular fibrillation and tachycardia.
(2) Auricular tachycardia, regular sinus rhythm, auricular premature systole.
(3) Auricular flutter with irregular conduction.
(4) Auricular flutter with variable block

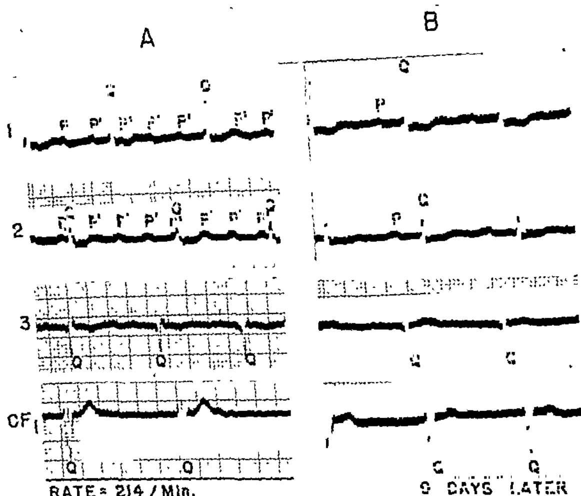


Figure 201 (Case No 8) Ectopic auricular tachycardia Auricular rate 214 P' wave upright in leads 1, 2 and 3 Ta waves absent High auricular ectopic focus Varying ventricular rates Ventricular response irregular Marked ventricular aberration

INTERPRETATION (A) AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK FROM HIGH AURICULAR FOCUS, AURICULAR RATE 214, VENTRICULAR ABERRATION PRESENT (B) REGULAR SINUS RHYTHM

Comment: The focus in this case is at or close to the sino-auricular node because the P' wave is identical to the P wave.

Electrocardiographers' Interpretation.

(1) Auricular tachycardia.
(2) Auricular tachycardia.
(3) Auricular tachycardia.
(4) Auricular flutter
(5) Auricular flutter

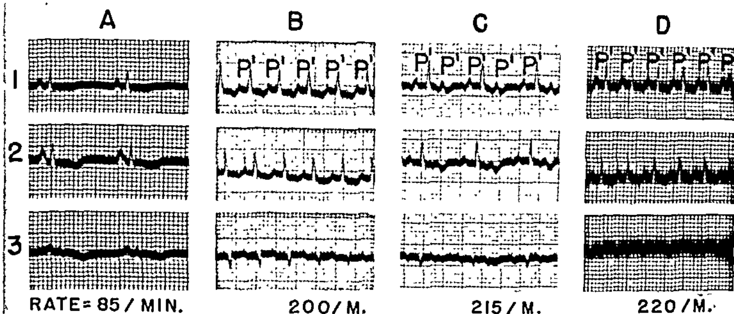


Figure 199 (Case No. 4) (A) Regular sinus rhythm with rate 85. P wave upright in leads 1, 2 and 3 P-R interval .10. Tracings in B, C and D are also from the same patient.

(B) Ectopic auricular tachycardia with auricular rate 200 P' wave upright in leads 1, 2 and 3. Ta waves masked in leads 1, 2 and 3 High auricular ectopic focus P'-R interval 12. Ventricular rate 200 Ventricular response regular with A V 1 1.

(C) Ectopic auricular tachycardia with auricular rate 215 P' waves same. Ventricular rate 107, with regular ventricular response with A V 2 1.

INTERPRETATION AURICULAR TACHYCARDIA FROM HIGH AURICULAR FOCUS IN WHICH THERE IS A 2 1 A-V BLOCK IN C AT RATE 215. THE BLOCK IS ABSENT IN B AND D AT RATES OF 200 AND 220.

Comment: It is tracings of this type that are very difficult to diagnose according to present day criteria.

Electrocardiographers' Interpretation

(1) Sinus rhythm, auricular tachycardia, auricular tachycardia with 2.1 block, auricular tachycardia

(2) Sinus rhythm, auricular tachycardia, auricular tachycardia with 2.1 block, auricular tachycardia.

(3) Sinus rhythm, auricular tachycardia, auricular tachycardia with 2.1 block, auricular tachycardia.

(4) Sinus rhythm, auricular flutter, auricular flutter with 2 1 block, auricular flutter.

(5) Sinus rhythm, auricular tachycardia, auricular tachycardia with 2:1 block, auricular tachycardia.

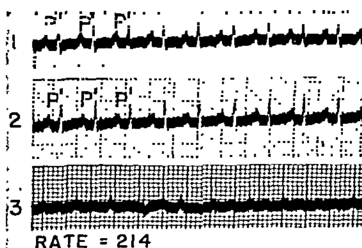


Figure 200 (Case No 5): Ectopic auricular tachycardia Auricular rate 214 P' wave upright in leads 1 and 2. Masked in lead 3 Ta waves masked in leads 1, 2 and 3 High auricular ectopic focus. P'-R interval 12 Ventricular rate 214 Ventricular response regular A V 1.1.

INTERPRETATION AURICULAR TACHYCARDIA FROM HIGH AURICULAR FOCUS WITH AURICULAR RATE 214

Electrocardiographers' Interpretation

(1) Auricular tachycardia.
(2) Auricular tachycardia
(3) Auricular tachycardia.
(4) Supraventricular tachycardia.
(5) Auricular tachycardia

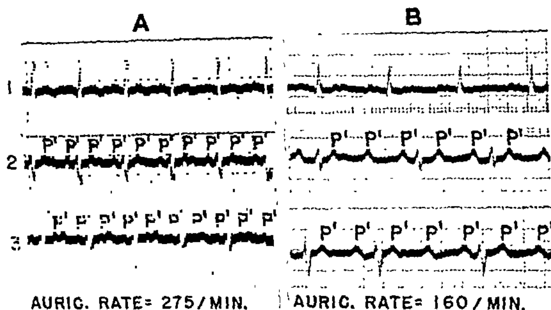


Figure 203 (Case No 8): Ectopic auricular tachycardia Auricular rate 275. P' wave upright in leads 2 and 3, absent in lead 1. Ta waves absent. High auricular ectopic focus Ventricular rate 137. Ventricular response regular A.V 2.1. Slight ventricular aberration

INTERPRETATION: (A) AURICULAR TACHYCARDIA WITH 2 1 A-V BLOCK, HIGH AURICULAR FOCUS, AURICULAR RATE 275 (B) SAME PATIENT AFTER QUINIDINE: AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK FROM HIGH AURICULAR FOCUS, AURICULAR RATE 160.

Electrocardiographers' Interpretation:

(1) Auricular tachycardia
(2) Auricular flutter.
(3) Auricular tachycardia
(4) Auricular flutter.
(5) Auricular tachycardia.

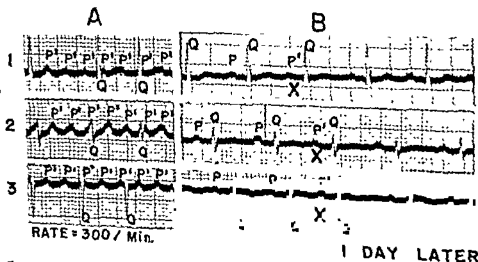


Figure 204 (Case No 9) Ectopic auricular tachycardia Auricular rate 300. P' wave upright in leads 1, 2 and 3 Ta waves absent High auricular ectopic focus Ventricular rate variable Ventricular response irregular Marked ventricular aberration

INTERPRETATION

IHC ... BLOCK FROM
LAI ... (B) REGU-

...tation:

(1) Auricular flutter
(2) Auricular flutter
(3) Auricular flutter
(4) Auricular flutter.
(5) Auricular tachycardia.

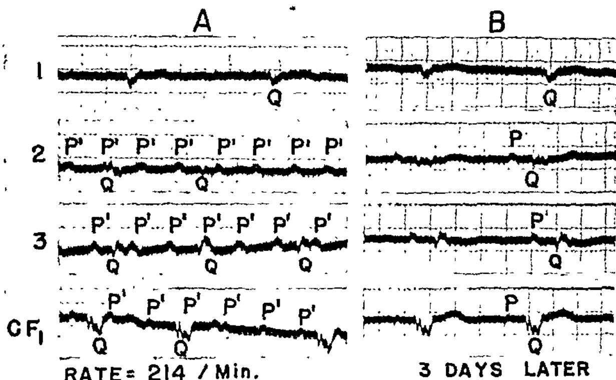


Figure 202 (Case No. 7): Ectopic auricular tachycardia. Auricular rate 214 P' wave upright in leads 2, 3 and CF₁ Absent in lead 1. Ta waves absent. High auricular ectopic focus *Varying ventricular rate Ventricular response* irregular. Marked ventricular aberration

INTERPRETATION: (A) AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK FROM HIGH AURICULAR FOCUS AT OR NEAR THE SINUS NODE, AURICULAR RATE 214. (B) REGULAR SINUS RHYTHM

Electrocardiographers' Interpretation.

(1) Auricular tachycardia
(2) Auricular tachycardia.
(3) Auricular flutter.
(4) Auricular flutter.
(5) Auricular flutter.

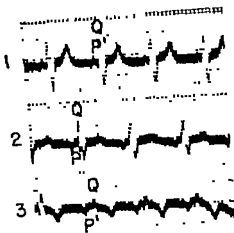


Figure 206 (Case No 11) Ectopic auricular tachycardia Auricular rate 115 P' wave upright in lead 1, inverted in leads 2 and 3 Ta waves absent Low auricular ectopic focus P'-R interval .08 Ventricular rate 115 Ventricular response regular A-V 1 1

INTERPRETATION· AURICULAR TACHYCARDIA FROM LOW AURICULAR FOCUS, AURICULAR RATE 115

Comment Auricular tachycardia with this short P'-R interval is often diagnosed high nodal tachycardia however, such records can be produced experimentally by stimulation of the caudal region of the human auricle remote from auriculo-ventricular node

Electrocardiographers' Interpretation
(1) Nodal rhythm
(2) Nodal rhythm
(3) Nodal rhythm
(4) Nodal rhythm
(5) Nodal rhythm

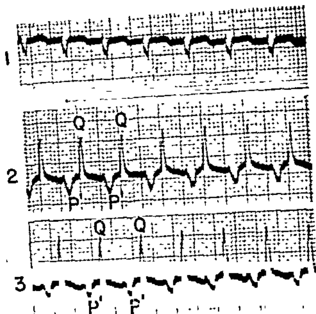


Figure 207 (Case No 12). Ectopic auricular tachycardia. Auricular rate 150 P' wave inverted in leads 2 and 3. Absent in lead 1. Ta waves absent. Low auricular ectopic focus P'-R interval .18 Ventricular rate 150. Ventricular response regular A V 1.1.

INTERPRETATION: AURICULAR TACHYCARDIA FROM LOW AURICULAR FOCUS, WITH RATE 150.

Electrocardiographers' Interpretation.
(1) Auricular tachycardia.
(2) Auricular tachycardia
(3) Nodal tachycardia.
(4) Nodal tachycardia.
(5) Low auricular or nodal tachycardia

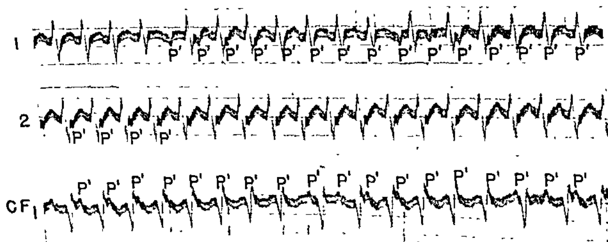


Figure 208 (Case No 13) Ectopic auricular tachycardia Auricular rate 187 P' wave inverted in leads 1 and 2, upright in CF_1 Ta waves absent Low auricular ectopic focus Varying ventricular rate Ventricular response irregular, marked ventricular aberration

INTERPRETATION AURICULAR TACHYCARDIA WITH PROGRESSIVE A-V BLOCK OF THE WENCKEBACH TYPE FROM LOW AURICULAR FOCUS, AURICULAR RATE 187. VENTRICULAR ABERRATION PRESENT

Electrocardiographers' Interpretation.
(1) Auricular tachycardia with Wenckebach
(2) Parasystole or flutter with A-V dissociation.
(3) Auricular tachycardia with Wenckebach.
(4) Auricular tachycardia with Wenckebach.
(5) Auricular flutter with Wenckebach.

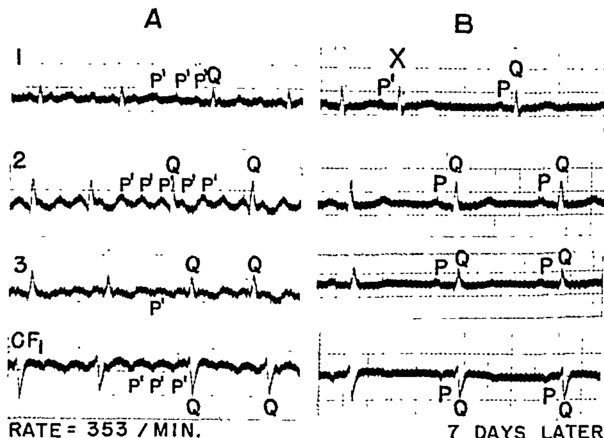


Figure 205 (Case No 10): Ectopic auricular tachycardia. Auricular rate 353. P' wave upright in 1, probably upright in 2, inverted in leads 3 and CF_1. Ta waves absent. High auricular ectopic focus. Ventricular rate varying. Ventricular response irregular. Marked ventricular aberration.

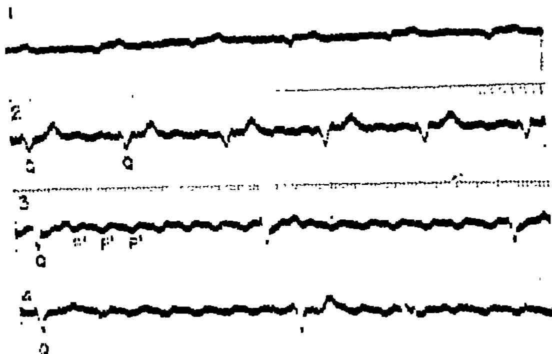
INTERPRETATION (A) AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK FROM HIGH AURICULAR FOCUS, AURICULAR RATE 353.

(B) REGULAR SINUS RHYTHM WITH AURICULAR PREMATURE SYSTOLE FROM SAME FOCUS.

Comment. In lead 2 the flutter deflections are of the sine wave type.

Electrocardiographers' Interpretation.

(1) Auricular flutter.
(2) Auricular flutter.
(3) Auricular flutter.
(4) Auricular flutter.
(5) Auricular flutter.



AURIC. RATE = 200 / Min.

Figure 211 (Case No 16) Ectopic auricular tachycardia Auricular rate 200 P' wave inverted in lead 3 absent in leads 1 and 2 Ta waves absent Low ectopic auricular focus Ventricular rate varying Ventricular response irregular Marked ventricular aberration.

INTERPRETATION AURICULAR TACHYCARDIA WITH COMPLETE HEART BLOCK FROM LOW ECTOPIC AURICULAR FOCUS, AURICULAR RATE 214

Electrocardiographers' Interpretation

(1) Auricular tachycardia with complete heart block with idioventricular rhythm
(2) Auricular flutter with variable A-V block and probable right bundle-branch block
(3) Auricular flutter.
(4) Auricular flutter.
(5) Auricular flutter with variable block

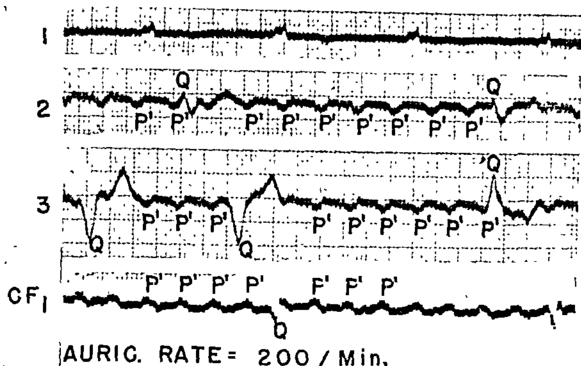


Figure 209 (Case No. 14). Ectopic auricular tachycardia Auricular rate 200. P' wave upright in CF_1. Inverted in leads 2 and 3 Absent in lead 1. Ta waves absent Low auricular ectopic focus. Varying ventricular rate Ventricular response irregular. Marked ventricular aberration.

INTERPRETATION AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK FROM LOW ECTOPIC AURICULAR FOCUS, AURICULAR RATE 200 HOWEVER, THIS MIGHT BE COMPLETE A-V BLOCK WITH IDIOVENTRICULAR RHYTHM OR PREMATURE VENTRICULAR SYSTOLES

Electrocardiographers' Interpretation

(1) Auricular tachycardia with complete heart block and ventricular pacemaker from more than one focus
(2) Auricular flutter with left bundle-branch block and ventricular ectopic beat.
(3) Auricular flutter.
(4) Auricular flutter with aberration and ventricular ectopic beats.
(5) Auricular tachycardia with a variable bundle branch block.

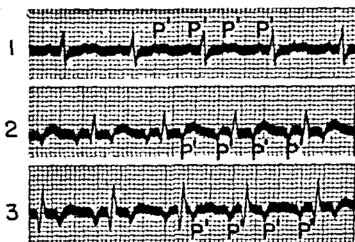
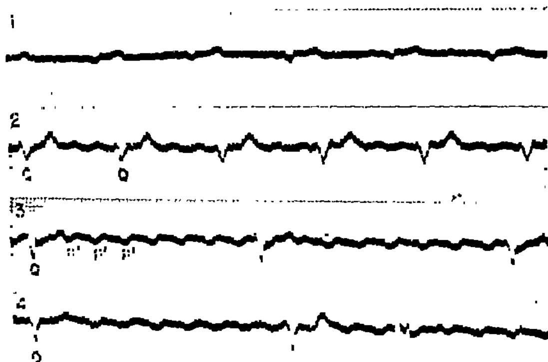


Figure 210 (Case No. 15) Ectopic auricular tachycardia with premature ventricular systoles Auricular rate 200 P' wave upright in lead 1, inverted in leads 2 and 3 Ta waves absent Low auricular ectopic focus P'-R interval 12 Ventricular rate 100 Ventricular response irregular A V 2.1

INTERPRETATION AURICULAR TACHYCARDIA WITH A-V BLOCK 2 1 FROM LOW ECTOPIC AURICULAR FOCUS, AURICULAR RATE 200

Electrocardiographers' Interpretation

(1) Auricular tachycardia with 2 1 A-V block
(2) Auricular flutter with 2 1 A-V block
(3) Nodal tachycardia with 2 1 A-V block
(4) Auricular flutter with 2 1 A-V block
(5) Auricular flutter or nodal tachycardia with 2 1 A-V block



AUR. C. RATE = 200 / Min.

Figure 211 (Case No. 16). Ectopic auricular tachycardia Auricular rate 200 P' wave inverted

uicular aberration

INTERPRETATION AURICULAR TACHYCARDIA WITH COMPLETE HEART BLOCK FROM LOW ECTOPIC AURICULAR FOCUS, AURICULAR RATE 214

Electrocardiographers' Interpretation

(1) Auricular tachycardia with complete heart block with idioventricular rhythm.
(2) Auricular flutter with variable A-V block and probable right bundle-branch block
(3) Auricular flutter
(4) Auricular flutter.
(5) Auricular flutter with variable block.

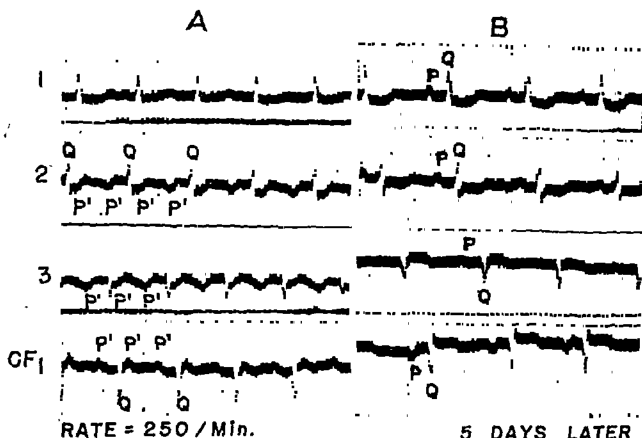


Figure 212 (Case No 17) Ectopic auricular tachycardia. Auricular rate 250 P' wave upright in leads 1 and CF_1, inverted in leads 2 and 3. Ta waves absent. Low auricular ectopic focus. Ventricular rate 125. Ventricular response regular A V 2.1.

INTERPRETATION. (A) AURICULAR TACHYCARDIA WITH 2.1 A-V BLOCK FROM LOW AURICULAR ECTOPIC FOCUS, AURICULAR RATE 250. (B) REGULAR SINUS RHYTHM.

Electrocardiographers' Interpretation

(1) Auricular *flutter* with 2.1 block.
(2) Auricular flutter with 2.1 block.
(3) Auricular flutter with 2:1 block.
(4) Auricular flutter with 2.1 block.
(5) Auricular flutter with 2.1 block.

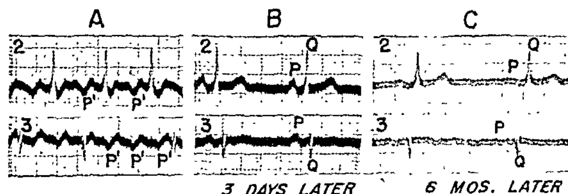


Figure 213 (Case No 18). Ectopic auricular tachycardia Auricular rate 250 P' waves inverted in leads 2 and 3. Ta wave upright in leads 2 and 3 Low auricular ectopic focus Ventricular rate varying Ventricular response irregular. Marked ventricular aberration

INTERPRETATION (A) AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK FROM LOW AURICULAR FOCUS, AURICULAR RATE 250 MARKED VENTRICULAR ABERRATION (B AND C) REGULAR SINUS RHYTHM

Comment Three days later regular sinus rhythm with P waves of increased amplitude Six months later regular sinus rhythm with P waves becoming smaller We have frequently noted this increased amplitude of P waves after the termination of tachycardia This increased amplitude may remain present for several days

Electrocardiographers' Interpretation

(1) Auricular flutter
(2) Auricular flutter
(3) *Auricular flutter*
(4) Auricular flutter.
(5) Auricular flutter

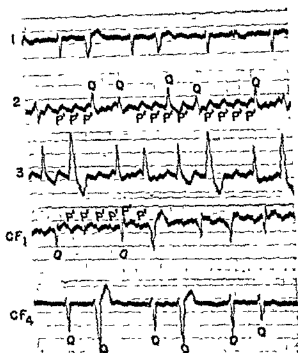


Figure 214 (Case No 19) Ectopic auricular tachycardia Auricular rate 273 P' wave inverted in leads 2 and 3, absent in leads 1 and CF_1, diphasic in CF_4 Ta waves upright in leads 2 and 3 Masked in leads 1, CF_1 and CF_4. Low auricular ectopic focus. Varying ventricular rate Ventricular response irregular Marked ventricular aberration

INTERPRETATION AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK FROM LOW AURICULAR FOCUS, AURICULAR RATE 273 VENTRICULAR ABERRATION PRESENT

Comment The bizarre ventricular complexes in lead 3 are probably due to ventricular aberration because of the intermediate degrees of aberration in the intervening complexes They are not ventricular premature systoles

Electrocardiographers' Interpretation

(1) Auricular flutter with variable block
(2) Auricular flutter with variable block
(3) Auricular flutter
(4) Auricular flutter
(5) Auricular flutter

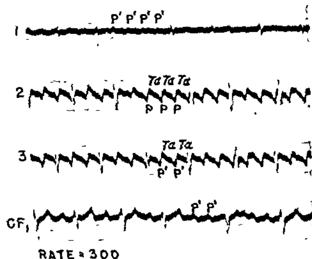


Figure 215 (Case No 20). Ectopic auricular tachycardia. *Auricular rate 300* P' waves upright *in leads 1 and* CF_1, inverted in leads 2 and 3 Ta waves upright in leads 2 and 3, absent *in leads 1 and* CF_1 Low auricular ectopic focus. Varying ventricular rate. Irregular ventricular response. Marked ventricular aberration.

INTERPRETATION· AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK FROM LOW AURICULAR ECTOPIC FOCUS, AURICULAR RATE 300. VENTRICULAR ABERRATION PRESENT.

Electrocardiographers' Interpretation.

(1) Auricular flutter with irregular block.
(2) Auricular flutter with irregular block.
(3) Auricular flutter with irregular block and variable flutter rate.
(4) Auricular flutter
(5) Auricular flutter

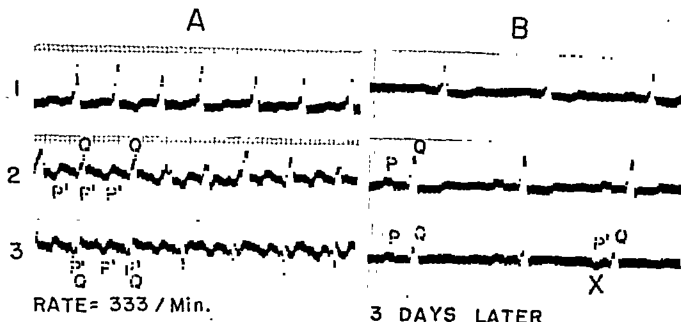


Figure 216 (Case No. 21). Ectopic auricular tachycardia. Auricular rate 333. P' waves inverted in leads 2 and 3, absent in lead 1. Ta waves upright in leads 2 and 3, absent in lead 1. Low auricular ectopic focus. Ventricular rate varying Irregular ventricular response. Marked ventricular aberration

INTERPRETATION (A) AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK FROM LOW AURICULAR ECTOPIC FOCUS, AURICULAR RATE 333 VENTRICULAR ABERRATION PRESENT (B) REGULAR SINUS RHYTHM WITH AURICULAR PREMATURE SYSTOLE FROM SAME ECTOPIC FOCUS

Electrocardiographers' Interpretation·

(1) Auricular flutter with variable block.
(2) Auricular flutter with variable block
(3) Auricular flutter.
(4) Auricular flutter
(5) Auricular flutter.

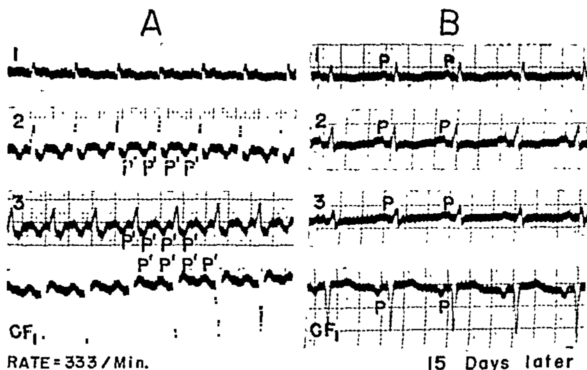


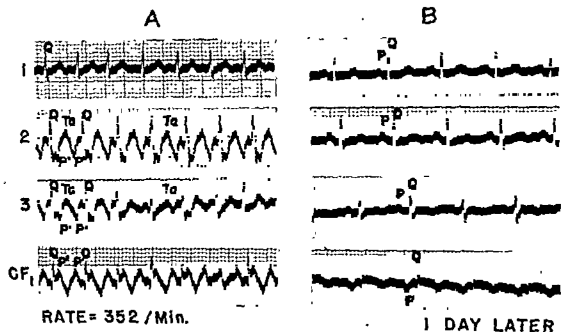
Figure 217 (Case No. 22) Ectopic auricular tachycardia Auricular rate 333 P' waves upright in leads CF_1, inverted in 2 and 3 and masked in lead 1 Ta waves absent in leads 1, 2 and 3, inverted in CF_1 Low auricular ectopic focus Ventricular rate 166 Ventricular response regular A V 2 1 Slight ventricular aberration

INTERPRETATION (A) AURICULAR TACHYCARDIA WITH 2 1 A-V BLOCK FROM LOW AURICULAR ECTOPIC FOCUS, AURICULAR RATE 333. (B) REGULAR SINUS RHYTHM

Comment The change in shape of the QRS simulating ventricular aberration is probably due to axis shift

Electrocardiographers' Interpretation

(1) Auricular flutter with 2 1 block
(2) Auricular flutter with 2 1 block.
(3) Auricular flutter with 2.1 block
(4) Auricular flutter with 2 1 block
(5) Auricular flutter with 2 1 block.



[illegible] aberration

INTERPRETATION· (A) AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK FROM LOW AURICULAR ECTOPIC FOCUS, AURICULAR RATE 352 VENTRICULAR ABERRATION PRESENT. (B) REGULAR SINUS RHYTHM

Electrocardiographers' Interpretation·

(1) Auricular flutter with 2.1 block.
(2) Auricular flutter.
(3) Auricular flutter with aberrant A-V conduction
(4) Auricular flutter.
(5) Auricular flutter.

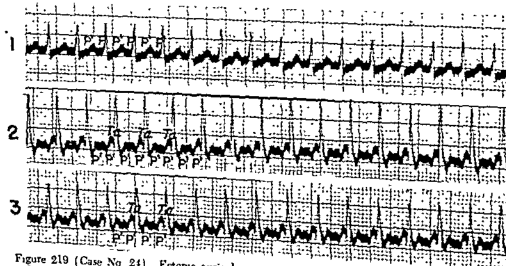


Figure 219 (Case No 24) Ectopic auricular tachycardia Auricular rate 400 P' waves are barely visible but probably upright in lead 1, inverted in leads 2 and 3 Ta waves upright in leads 2 and 3, absent in lead 1 Low auricular ectopic focus Ventricular rate 200 Ventricular response regular A V 2 1

INTERPRETATION AURICULAR TACHYCARDIA WITH 2 1 A-V BLOCK FROM LOW AURICULAR FOCUS, AURICULAR RATE 200.

Electrocardiographers' Interpretation:

(1) Auricular flutter with 2.1 block.
(2) Auricular tachycardia with 2:1 block.
(3) Supraventricular tachycardia with block.
(4) Auricular flutter with 2.1 block.

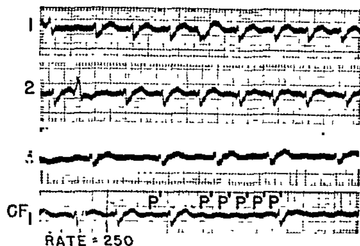


Figure 220 (Case No 25): Ectopic auricular tachycardia Auricular rate 250. P′ wave upright in CF_1, small in leads 1, 2 and 3. Ta waves absent Mid-auricular ectopic focus Varying ventricular rate. Irregular ventricular response. Marked ventricular aberration.

INTERPRETATION. AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK, ECTOPIC FOCUS IS IN THE "SILENT" AREA (MID-AURICULAR), AURICULAR RATE 250.

Comment· Premature ventricular systoles in lead 2 and not aberration In leads 1, 2 and 3, one could not differentiate this tracing from auricular fibrillation with bundle branch block.

Electrocardiographers' Interpretation.

(1) Auricular flutter with predominant 2·1 block and defective intraventricular conduction.
(2) Flutter-fibrillation.
(3) Auricular flutter
(4) Auricular fibrillation
(5) Auricular tachycardia with bundle branch block.

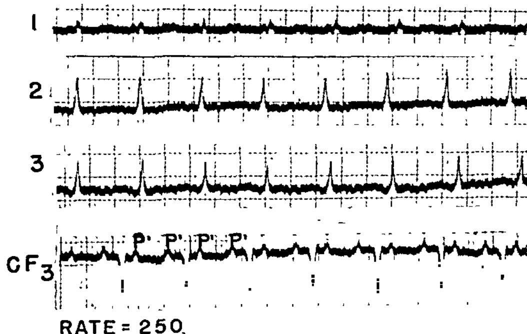


Figure 221 (Case No. 26) Ectopic auricular tachycardia Auricular rate 250 P′ wave upright in CF_3, undetermined in leads 1, 2 and 3 Ta waves absent. Mid-auricular ectopic focus. P′-R interval 16 Ventricular rate 125 Regular ventricular response

INTERPRETATION AURICULAR TACHYCARDIA WITH A-V BLOCK 2 1, ECTOPIC FOCUS "SILENT" AREA (MID-AURICULAR), AURICULAR RATE 250

Comment If not for the evidence of P′ waves in CF_3 this tracing would be called nodal tachycardia.

Electrocardiographers' Interpretation.

(1) Auricular tachycardia with 2 1 block.
(2) Nodal tachycardia with variation in A-V conduction
(3) Auricular tachycardia
(4) Low auricular or nodal tachycardia.
(5) Auricular tachycardia

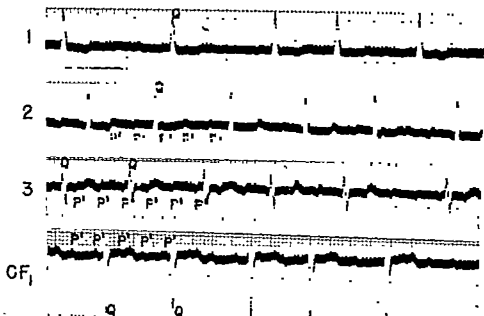


Figure 222 (Case No 27). Ectopic auricular tachycardia. Auricular rate 230. P' waves upright in lead CF_1. Biphasic in leads 2 and 3 and undetermined in lead 1 Ta waves absent. Mid-auricular ectopic focus Varying ventricular rate. Irregular ventricular response. Marked ventricular aberration

INTERPRETATION· AURICULAR TACHYCARDIA WITH VARYING A-V BLOCK, ECTOPIC FOCUS "SILENT" AREA (MID-AURICULAR), AURICULAR RATE 230 VENTRICULAR ABERRATION PRESENT

Electrocardiographers' Interpretation

(1) Auricular tachycardia with irregular block.
(2) Auricular flutter with variable A-V block.
(3) Auricular flutter with 3 1 block
(4) Auricular flutter
(5) Auricular flutter.

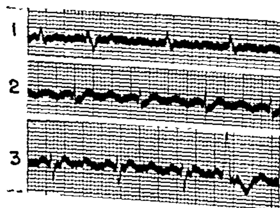


Figure 223 (Case No 28): Ectopic auricular tachycardia Auricular rate 250 P' wave undetermined in lead 1, sine wave in leads 2 and 3. Ta waves absent Undeterminable auricular focus Ventricular rate 88. Regular ventricular response A V 3 1 with premature ventricular systole in lead 3.

INTERPRETATION· AURICULAR TACHYCARDIA WITH 3 1 A-V BLOCK UNDETERMINABLE AURICULAR FOCUS, AURICULAR RATE 250.

Electrocardiographers' Interpretation

(1) Auricular flutter
(2) Auricular flutter.
(3) Auricular flutter
(4) Auricular flutter.
(5) Auricular flutter.

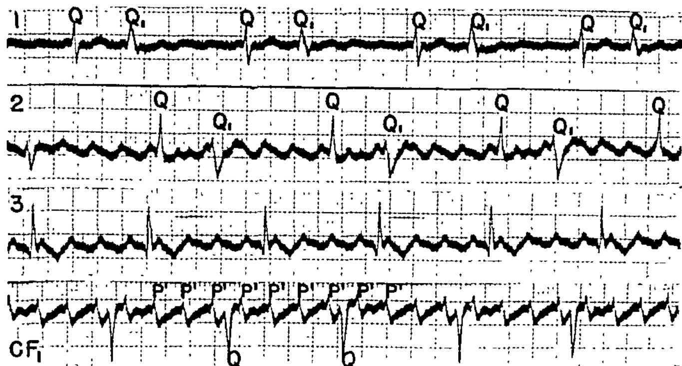


Figure 224 (Case No 29): Ectopic auricular tachycardia Auricular rate 250. P' waves are of the sine wave type in leads 1, 2 and 3. Ta waves are masked Ventricular rate varying. Irregular ventricular response.

INTERPRETATION AURICULAR TACHYCARDIA OF THE SINE WAVE TYPE WITH VARYING A-V BLOCK, ECTOPIC FOCUS UNDETERMINABLE, AURICULAR RATE 250

Comment: Couplings in leads 1 and 2, due to either premature ventricular systoles or to ventricular aberration It is impossible to differentiate which of the two is present. Note the distinct auricular intrinsicoid deflection in lead CF_1.

Electrocardiographers' Interpretation:

(1) Auricular flutter with predominant 4 1 block and premature ventricular systoles.
(2) Auricular flutter
(3) Auricular flutter with ventricular ectopic beats and aberrant A-V conduction
(4) Auricular flutter.
(5) Auricular flutter with variable block and ectopic ventricular beats.

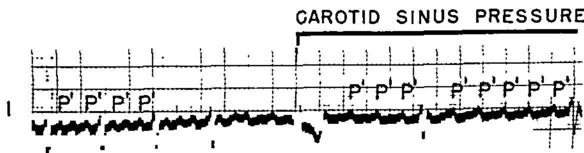


Figure 225 (Case No 30) Ectopic auricular tachycardia with premature ventricular systole Auricular rate 200 P' wave upright in lead 1 Ta wave upright in lead 1 Varying ventricular rate

INTERPRETATION AURICULAR TACHYCARDIA WITH A-V BLOCK 2 1 AURICULAR RATE 200

Comment In the first part of the tracing the auricular rate appears to be 400 because the T waves are confused with P' waves since they fall half way between P'-P' After carotid sinus pressure the true auricular rate is apparent

Electrocardiographers' Interpretation

(1) Auricular flutter with slowing of the auricular rate after carotid sinus stimulation
(2) Auricular flutter
(3) Auricular flutter.
(4) Auricular flutter
(5) Auricular flutter.

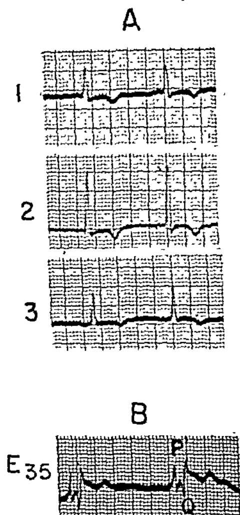


Figure 226 (Case No. 31). Ectopic auricular rhythm. P and Ta waves absent Mid-auricular ectopic focus. Ventricular rate 54

INTERPRETATION ECTOPIC AURICULAR RHYTHM FROM MID-AURICULAR FOCUS, AURICULAR RATE 54

Comment The standard limb leads might be interpreted as nodal rhythm which it obviously is not as shown by the esophageal tracing Ectopic focus is in the "silent" area of the auricle

Electrocardiographers' Interpretation
(1) Nodal rhythm
(2) Nodal rhythm
(3) Sinus or upper nodal rhythm

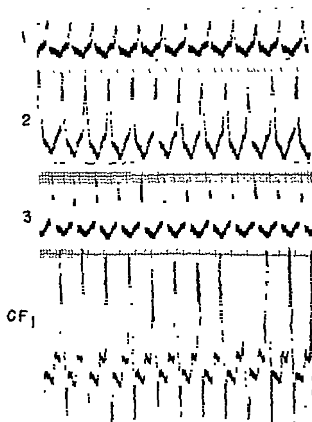


Figure 227 (Case No. 32). *Ectopic tachycardia* Auricular rate undeterminable. P' and Ta waves masked in leads 1, 2, 3 and CF_1 Ventricular rate 200. *Ventricular* response regular.

INTERPRETATION: ECTOPIC TACHYCARDIA, AURICULAR RATE ... ERMINABLE.

n:

(3) Supraventricular tachycardia.
(4) Auricular flutter.
(5) Auricular flutter

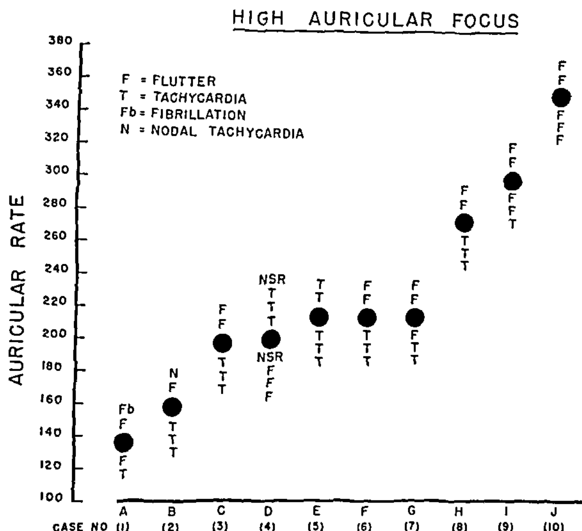


Figure 228. First 10 cases from high auricular focus arranged in order of increasing rates The *electrocardiographers' diagnoses are indicated* at each point In only two instances is there complete agreement Note that there is no *sharp line of demarcation between "tachycardia" and "flutter"*

SUMMARY AND CONCLUSION

Thirty-two electrocardiograms of the auricular arrhythmias have been presented, each accompanied by the interpretations of five leading electrocardiographers rendered according to standard present-day nomenclature together with a descriptive type of physiologic diagnosis proposed as a substitute for the current method of interpretation. Graphic analysis of the former diagnoses has demonstrated that current nomenclature is subject to wide variations and results in confusion since it is based on arbitrary criteria. It is apparent from the charts that no line of distinction exists between tachycardia and flutter.

In view of this fact, the verbal distinction between *"tachycardia" and "flutter" is misleading.* Such arbitrary distinctions are obviated when current nomenclature is replaced by a description of the *actual events occurring in the auricles.* Based on this principle of physiologic descriptive diagnosis suggested in Chapter IX, the auricular arrhythmia heretofore known as "auricular flutter" should be considered as a form of tachycardia, usually arising at the caudal end of the auricle, and occurring at an auricular rate rapid enough to cause the development of a physiologic auriculo-ventricular block.

Strictly speaking, the term "auricular flutter" should be abolished promptly. Almost inevitably, however, confusion would result from such an abrupt change in nomenclature. In deference to traditional usage and for purposes of clarity, therefore, we have used the term

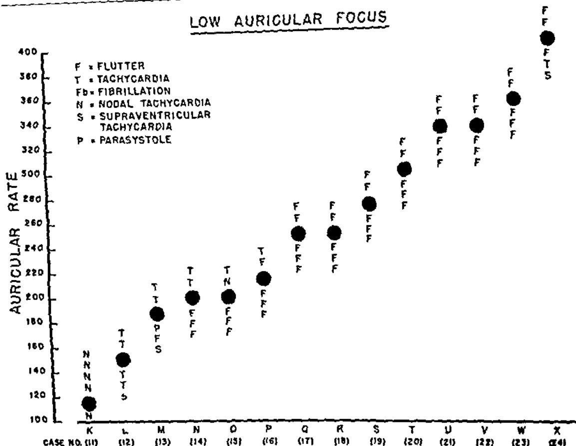


Figure 229 Fourteen cases from low auricular focus arranged in order of increasing rate. The electrocardiographers' diagnoses are indicated at each point. In only eight instances is there complete agreement. This chart clearly shows that there is no sharp line of demarcation between "tachycardia" and "flutter."

throughout the remainder of this monograph but with the implicit stipulation that auricular flutter is a tachycardia at an auricular rate rapid enough to cause physiologic auriculoventricular block. It is hoped that the designation "auricular flutter" gradually will fall into disuse as physicians and medical students become familiar with the physiologic descriptive nomenclature illustrated in the present chapter. Only when "tachycardia" and "flutter" are diagnosed in terms of the physiologic disturbance will the prevailing confusion be avoided and treatment placed on a rational and understandable basis.

CHAPTER XI

Clinical Aspects of Auricular Fibrillation

AURICULAR fibrillation is a cardiac arrhythmia consisting of asynchronous fibrillary twitchings in various parts of the auricular walls, to which the ventricles respond irregularly with contractions of variable force depending upon the integrity of the conducting tissue. Some of the ventricular beats are too weak to produce a palpable pulse, with the result that the pulse rate at the radial artery may be appreciably lower than the heart rate determined at the apex (pulse deficit). The cinematographic and electrocardiographic characteristics of auricular fibrillation are described in detail in Chapters XII, XIII and XIV.

Incidence and Etiology: Auricular fibrillation is among the most serious and frequent disorders of the cardiac rhythm, and is the commonest arrhythmia observed in decompensating hearts. Approximately 70 per cent of all cases of advanced failure display this form of arrhythmia at some time.[387] "Absolute arrhythmia" (pulsus irregularis absolutis), "irregular irregularity" and "delirium cordis" are synonyms indicating the essential clinical characteristics of the condition. The term "perpetual arrhythmia" (pulsus irregularis perpetuus), formerly used in the belief that fibrillation is a permanent disorder, was abandoned with the discovery that many patients suffer repeated attacks which terminate spontaneously (paroxysmal fibrillation). The majority of instances occur in association with rheumatic and hypertensive heart disease, in hyperthyroid states and in elderly persons. More rarely, fibrillation develops in young, healthy individuals with otherwise normal hearts. The incidence is greatest during the third and fourth decades among patients with valvular heart disease and during the sixth and seventh decades among those with nonvalvular heart disease. Although auricular fibrillation may develop at any age, it is relatively infrequent in the first two decades and is rarely seen in children under 12 years of age. Goldbloom and Segall[237] observed auricular fibrillation in a three-month old infant whose heart was otherwise normal, spontaneous conversion to sinus rhythm occurred at one year of age, and a 10 year follow-up study failed to reveal evidence of cardiac disease.

The sex incidence varies widely in the series reported to date but in general auricular fibrillation associated with rheumatic heart disease occurs more frequently among women while the arrhythmia accompanying degenerative heart disease is found more frequently in men. White[638] noted that twice as many instances of auricular fibrillation among males as among females; the converse was true among Cookson's[110] patients. Luten and Jeffries[403] found an almost equal sex distribution in their series of 431 cases. The small number of patients with fibrillation and heart disease described by Evans[175] included a slight predominance of women among the rheumatic group and a ratio of three men to each woman in the nonrheumatic group Of 119 cases of auricular fibrillation in nonrheumatic diseased hearts studied at autopsy by Brown,[72] approximately two-thirds were in males, however, 12 of the 15 hearts which showed no pathologic lesions were from females. Friedlander and Levine's[210] series of 35 instances of fibrillation without evidence of organic heart disease included 32 males. In a group of 651 cases reported by Hanson and

Rutledge[259] all instances of established fibrillation and 55 per cent of instances of paroxysmal fibrillation were in men

Auricular Fibrillation in Heart Disease: Auricular fibrillation may accompany any type of heart disease but is most frequently associated with rheumatic valvulitis, particularly mitral stenosis In a tabulation of 14 available reports covering a total of 2,958 cases of auricular fibrillation, Evans[173] found the incidence of rheumatic heart disease to vary from 20 to 69 per cent, averaging approximately 49 per cent Evans' own series of 39 patients with fibrillation included 22 examples of rheumatic heart disease (54 per cent).

Elderly patients with nonvalvular or nonrheumatic heart disease often exhibit auricular fibrillation.[72, 110, 343, 591] Authorities disagree concerning the exact nature of the pathologic process present in this group. Levine[343] described it as "the condition called chronic myocarditis in middle-aged or elderly people in whom there is no important disease of the valves but rather some functional or structural damage to the musculature of the heart" Cookson's[110] patients with nonvalvular heart disease and auricular fibrillation displayed "no constant associated conditions, except that of age" On the other hand, Brown's[72] study of 119 instances of this type revealed the presence of hypertension in 79 3 per cent of the patients with persistent fibrillation and in 86 5 per cent of those with transient fibrillation A pre-existing hypertension which may have disappeared with the onset of the arrhythmia or with the onset of congestive failure undoubtedly is responsible for certain instances of auricular fibrillation associated with nonvalvular heart disease In many such cases, however, some structural change of the myocardium associated with age, or "presbycardia" (a term suggested by Dock[145]), probably constitutes the underlying pathologic process

The majority of instances of advanced congestive failure are associated with auricular fibrillation. In all forms of heart disease, regardless of cause, the one factor most apt to predispose to the development of auricular fibrillation is congestive failure. Conversely, prolonged rapid fibrillation, like uncontrolled tachycardia, may precipitate cardiac decompensation. Thus, in a given instance it may be difficult to determine whether the arrhythmia or the congestive failure was the initial disturbance.

The common occurrence of auricular fibrillation in thyrotoxicosis has been noted by many observers.[5, 22, 32, 154, 230, 257, 292, 307, 324, 342] In 2,958 cases of auricular fibrillation which Evans[173] collected from the literature, the incidence of thyroid disease reported by various authors ranged between 0 and 44 per cent, averaging about 9 per cent. Parkinson and Campbell's[493] series of 200 examples of paroxysmal fibrillation included 28 cases (14 per cent) associated with goiter. The nature of the injury suffered by the heart in thyrotoxicosis remains obscure. Most authorities believe no permanent cardiac damage results even when extreme hyperthyroidism has existed over a prolonged period. A comprehensive review of this subject has been written by Maher and Sittler.[429] In a series of 180 cases these authors found that "congestive heart failure was not present in uncomplicated thyrotoxicosis The presence of this phenomenon was associated with the coexistence of a structural lesion. Abnormal cardiac physiologic function as determined clinically or demonstrated objectively by the electrocardiogram, such as heart block (all types), damage to the ventricular conduction system and auricular fibrillation, appeared to be manifestations of primary organic heart disease modified by the element of thyrotoxicosis. The effect of thyrotoxicosis appeared to be that of a catalytic agent. The course of the organic heart disease progressed more rapidly when thyrotoxicosis was active. The thyrotoxicosis brought to the surface latent cardiovascular lesions, which resumed their latency on the successful termination of the thyroid toxemia"

Syphilitic heart disease seldom is associated with auricular fibrillation Among 2,958 cases of fibrillation collected from the literature by

Evans[175] the incidence of syphilis was approximately 1.5 per cent; none of Evans' own series of 39 patients with persistent fibrillation exhibited the infection. Parkinson and Campbell[483] found syphilitic aortitis in five of 200 instances of paroxysmal fibrillation (2.5 per cent). In syphilitic heart disease, as in other types of cardiac conditions, the incidence of fibrillation is greater in the presence of congestive failure.

The association of angina pectoris with auricular fibrillation is likewise uncommon. Cookson[110] found only five such instances in a series of 2,000 patients. The rarity with which angina pectoris is observed in auricular fibrillation tends to support the conclusion of Brown;[72] Cookson;[110] and Brill and Meissner;[65] that coronary disease is not a significant etiologic factor in this arrhythmia. Nevertheless, the transient occurrence of fibrillation during the early course of acute coronary thrombosis has been noted frequently.[68, 345]

Both auricular fibrillation and subacute bacterial endocarditis are common complications in rheumatic heart disease, but the coincidental occurrence of these two conditions is comparatively rare. High-grade mitral stenosis appears to favor the development of fibrillation; bacterial endocarditis tends to be engrafted upon a mildly affected mitral valve and upon a deformed aortic valve[213] Cookson[110] noted only three instances (0.25 per cent incidence) of infective endocarditis in a series of 1,200 cases of auricular fibrillation. Two of these three patients came under observation with both conditions present; in the third, fibrillation developed immediately before death Cookson could "find no example in the literature in which the infection has developed in a patient already under observation with auricular fibrillation." Since the publication of this statement such instances have been noted by several observers[389] including the authors.

The existence of an etiologic relationship between auricular distention and auricular fibrillation is suggested by the frequent association of the arrhythmia with mitral stenosis, hypertension and congestive failure. This question has received the attention of several investigators[259, 403, 464] and is considered in Chapter XIV.

Auricular Fibrillation in Normal Hearts: The demonstration in 1913 by Gossage and Hicks[243] that auricular fibrillation may occur in an otherwise normal heart has been confirmed by numerous investigators.[66, 69, 203, 210, 343, 477, 483] A comprehensive review of the subject was recently presented by Hanson and Rutledge[259] together with a report of 30 such cases found among a series of 651 patients with fibrillation observed at the Lahey Clinic. On the basis of the various series reported, including their own, Hanson and Rutledge estimate that 6.5 per cent of all instances of fibrillation occur in normal hearts.

Effect of Drugs and Chemicals on the Induction of Auricular Fibrillation: As primary factors in the causation of clinical auricular fibrillation, drugs and chemicals cannot be considered of great importance. Orgain, Wolff and White[477] collected from the literature instances in which the development of fibrillation in normal hearts was attributed to epinephrine, digitalis, acetylsalicylic acid, ether, alcohol, tobacco, hydrogen sulphide, arsenic, carbon monoxide, and gasoline fumes. These rare occurrences, however, rather than constituting illustrations of specific pharmacologic action, may more logically be considered either as isolated instances of drug idiosyncrasy or as examples of the "trigger" mechanism suggested by Friedlander and Levine.[210] According to the latter concept, the drug or chemical acts as a trigger upon a heart which, though structurally normal, is functionally so altered as to be subject to the sudden inception of auricular fibrillation upon contact with some irritation or exciting agent. The same mechanism was suggested as a possible explanation of the spontaneous occurrence of fibrillation in certain individuals with otherwise normal hearts In such cases "it is possible that an inherent susceptibility to functional nervous instability provides a fertile

field for the propagation and persistence of aberrant stimuli whenever these people are subjected to a particular inciting factor; i.e., emotional strain, acute febrile or toxic disorders . . . the trigger may be related to the adrenal gland."[210]

As secondary factors in initiating fibrillation in definite disease states, the role of certain drugs and chemicals appears more clearly established. McEachern and Baker[407] induced fibrillation by the administration of digitalis in nine patients with cardiovascular syphilis; this finding is particularly significant in view of the comparative rarity with which the arrhythmia is observed in syphilitic heart disease. Tung[604] reported the appearance of transient auricular fibrillation in 15 patients in association with full or excessive digitalization, and disappearance of the arrhythmia on withdrawal of the drug Twelve of these patients presented gross evidence of heart disease Of the remainder, one suffered from mitral valvular obstruction due to a myxoma of the left atrium and another from severe toxemia resulting from suppurative pneumococcal pleurisy The fifteenth patient was a 64 year old man exhibiting carcinoma of the esophagus, atelectasis of the left lung and displacement of the heart to the left.

Rosenblum, Hahn and Levine[520] produced transient auricular fibrillation in hyperthyroid animals by the injection of doses of epinephrine too small to alter the cardiac rhythm of animals with normal or subnormal thyroid function Nahum and Hoff[464] converted normal heart action to auricular fibrillation in four patients with hyperthyroidism by administration of 0 75 mg of acetyl-B methylcholine chloride per kilogram of body weight but failed to induce such changes in normal persons given even larger doses of the drug Acetyl-B-methylcholine chloride also precipitated the arrhythmia in electrically shocked animals Since the principal action of this drug is vagus stimulation, Nahum and Hoff concluded that the production of fibrillation in their subjects was the effect of two distinct factors (1) a vagus factor (from the action of the drug); and (2) an exciting agent acting on the heart (thyroxine or electric shock), designated as the "E factor." They further theorized that the mechanism of clinical fibrillation involves the interplay of these two factors. The "E factor" is represented by the stretching of the auricular musculature as in mitral stenosis and in congestive failure, by the overactivity of the thyroid in thyrotoxicosis, and by functional nervous instability in some organically normal hearts. In the presence of any of the above states ("E factors"), should overactivity of the vagus develop or should the individual be abnormally vago-sensitive, auricular fibrillation may result. This theory is not incompatible with the "trigger" concept of Friedlander and Levine.[210]

In summary, the evidence presented in the literature to date fails to establish any single factor as a sufficient cause of auricular fibrillation. Rather, the development of the arrhythmia apparently depends on the interplay of a number of circumstances which most often arise in the presence of grave heart disease, especially during the stage of congestive failure, in certain toxic states, particularly thyrotoxicosis, and in elderly persons with or without heart disease. These circumstances, however, may also combine to initiate the arrhythmia in younger, healthy individuals with hearts which otherwise appear normal. The latter group includes probably some 4 to 9 per cent of all instances of auricular fibrillation.

Pathology: General agreement prevails among authorities that no specific histologic lesions are characteristic of auricular fibrillation. Yater[691] reached this conclusion after a comprehensive review of the literature and a microscopic study of 29 hearts which had been the seat of the arrhythmia. Similar conclusions were reached earlier by Frothingham;[211] and later by Cookson;[110] Lewis,[387] White;[638] Brown,[72] Stroud, Laplace and Reisinger;[591] and Friedlander and Levine.[210]

Duration of Auricular Fibrillation: Fibrillation is classified as (1) persistent, permanent,

or established; and (2) transient or paroxysmal. Both varieties may occur in association with any type of heart disease or in a heart which is otherwise normal. The transient variety may, after a number of paroxysms, become established. Few statistics are available concerning the relative frequency of the two types. In White and Jones'[638] series of 376 cases of fibrillation including 30 patients without other evidence of heart disease, 309 (82.2 per cent) were classified as established and 67 (17.8 per cent) as paroxysmal. In Kohn and Levine's[313] group of 49 patients including three in which no other heart disease was demonstrable, 46 (93.9 per cent) were classified as exhibiting permanent fibrillation.

Friedlander and Levine[210] "arbitrarily" classified fibrillation as permanent if the arrhythmia persisted longer than seven days, on the assumption that "any attack lasting more than a week would not be likely to cease spontaneously." In a series of 200 cases of fibrillation reported by Parkinson and Campbell,[483] the duration of the paroxysms varied from one to 48 hours in 160 patients, from two to four days in 20 patients, from four to seven days in eight patients and from seven days to a maximum of three weeks in the remaining 12 patients. Among Hanson and Rutledge's[259] patients with otherwise normal hearts 90 per cent of the paroxysms lasted less than 24 hours and 60 per cent less than four hours. Orgain, Wolff and White[477] describing their series of 49 cases, noted "great variation in the duration of paroxysms — from the shortest of a few minutes to the longest lasting one year." Usually, however, the paroxysms lasted "a few hours or days."

The above observations appear to justify the conclusion that in the large majority of cases of transient fibrillation the paroxysms do not last longer than seven days. Such cases have been observed by Burch,[77] Fogel,[109] and the authors.

Symptomatology: Palpitation is the most common symptom of auricular fibrillation. Exhaustion, breathlessness, faintness and giddiness may also accompany the arrhythmia. Pain is rarely a complaint. Orthopnea was the first symptom reported by 2 of our patients. The onset of the attack may not be associated with any particular subjective phenomena, so that the patient is unaware of the change in cardiac rhythm. In rare instances the onset is stormy, being accompanied by intense distress with symptoms of shock and prostration. Typically, however, the patient experiences the sudden onset of palpitation and a variable degree of discomfort without complete disability.

Diagnosis: Diagnosis rarely offers appreciable difficulty. In most cases the disturbance can be identified by means of the palpating finger and stethoscope (or naked ear). Sir Thomas Lewis[356] gives the following rule: "Given a decompensated cardiac with a heart rate as counted at the apex of over a hundred and a radial pulse that is appreciably less (a pulse deficit of ten or more beats) if the rhythm seems grossly irregular and the pulse is irregular in both time and force the condition is auricular fibrillation nine times out of ten."

Frequent, irregularly spaced extrasystoles occasionally pose a problem in differential diagnosis. In that condition, as in auricular fibrillation, short, weak beats are followed by compensatory long pauses. In fibrillation, however, long pauses also occur frequently after strong beats, a phenomenon not seen in the extrasystolic arrhythmia. Any factor which accelerates the pulse rate, such as exercise or atropine, tends to increase the arrhythmia of fibrillation and to diminish the irregularity due to extrasystoles; agents which lower the pulse rate have the converse effects. Finally, the diagnosis can be established conclusively by graphic methods. The disappearance of the auricular wave in polygraphic tracings rules out the presence of premature auricular systole. Electrocardiographically, auricular fibrillation is demonstrated either by the absence of P' waves and/or by a succession of arrhythmic oscillations of variable configuration and amplitude (see Chapter XIII). The ventricular complexes of fibrillation are irregularly spaced, but are otherwise normal in shape and size in a given trac-

ing except for changes due to possible associated disease involving either ventricle. Some degree of ventricular aberration often is present.

Effect of Auricular Fibrillation on Cardiac Function: The effect of fibrillation on cardiac function varies greatly. In some instances, prolonged fibrillation appears to cause little or no harm, especially if the heart action is maintained at a reasonably slow rate either by administration of digitalis or by organic heart block without drugs.[639] Friedlander and Levine[210] observed an instance of auricular fibrillation known to have persisted for 31 years without medication, the patient exhibited no incapacity until five years prior to death when a left homonymous hemianopsis developed due to cerebral embolism. Another patient described by these workers "has remained well for years without medication although the rate is always rapid." We have observed five siblings (two brothers and three sisters) all of whom exhibited fibrillation since childhood and none of whom ever required medical treatment of the arrhythmia At present, two of the sisters are alive at the ages of 74 and 82 years respectively. One sister died at the age of 78 and a brother died at the age of 75 The remaining brother died of coronary thrombosis and myocardial infarction (established at autopsy) at the age of 58 years. Similar instances of persistent "familial" fibrillation have been reported by Wolff,[674] and Hanson and Rutledge[259]

In other instances, grave disturbance in cardiac function rapidly follows the onset of fibrillation, although no structural changes of the heart may be demonstrable by available clinical means Such a case was observed[66, 67, 69] in a previously healthy woman. Fibrillation first appeared at the age of 43 years without apparent cause. After about three months without treatment, severe congestive failure developed with generalized edema and signs of fluid in the serous cavities Rest and digitalization for one week reduced the heart rate from 160 to 85, produced extensive diuresis with a loss of 15 pounds in weight, and restored compensation to a considerable extent. Roentgen-ray examination disclosed that the pleural and pericardial effusions had largely disappeared and the cardiac shadow was nearly normal. Electrocardiograms showed the auricles were still fibrillating but the heart was otherwise normal except for slight changes due to digitalis. A "test" dose of 3 grains (0.2 gram) of quinidine was administered, two hours later the heart rhythm was found to be normal. Quinidine in half grain doses twice daily was continued for two days, after which medication was discontinued. Within one month the patient resumed her normal mode of life. To this writing, 15 years later, she has remained entirely well. As well as can be determined by clinical methods, including radiographic and electrocardiographic studies, her heart is entirely normal. A similar example of severe cardiac dysfunction following the onset of auricular fibrillation is Case 186 reported by Parkinson and Campbell.[483] These authors point out "how much disability may be produced by fibrillation, apart from any other diseases of the heart."

Between the two extremes described above are found the majority of instances of fibrillation. In these, the arrhythmia causes a degree of cardiac embarrassment which adds measurably to the distress already present in patients with organic heart disease and which produces breathlessness and palpitation of variable severity in persons whose hearts are otherwise normal. On the other hand, for reasons not altogether clear, the presence of auricular fibrillation appears to reduce the possibility of the development of subacute bacterial endocarditis and to mitigate to some extent the pain of angina pectoris.[110]

The generally accepted conclusion that auricular fibrillation predisposes to the formation of auricular thrombi[313] notwithstanding, the incidence of embolism is not as much greater in this arrhythmia than in normal sinus rhythm as was formerly believed.[110, 313, 356] Embolism does occur more frequently, however, when the cardiac rhythm becomes normal after prolonged fibrillation. Apparently the sudden on-

set of normal rhythm is an important factor in the production of emboli.[313, 417]

Prognosis: Auricular fibrillation in the absence of other evidence of heart disease presents a favorable prognosis. Orgain, Wolff and White[477] found the few cardiovascular changes in their series of 49 such cases "rarely serious and in some instances wholly incidental to the presence of fibrillation. Hyperthyroidism was an uncommon complication. The mortality from auricular fibrillation or from cardiac disease was almost negligible." These conclusions are in agreement with those of Parkinson and Campbell;[483] Friedlander and Levine;[210] Willius and Dry;[654] and Cooke and White.[103] The example of familial fibrillation reported earlier in this chapter is further evidence of the relative benignancy of this arrhythmia occurring in an otherwise normal heart.

CHAPTER XII

The Motion of the Auricles In Auricular Fibrillation

Because of the rapidity and complexity of mechanical and electrical events during auricular fibrillation, investigation of this disturbance is more difficult than are parallel studies of auricular premature systole and tachycardia. Despite these inherent limitations, the application of modern equipment and techniques has yielded new evidence concerning the nature of auricular fibrillation. By means of high-speed motion pictures magnified to reveal the minutest details of auricular activity, the motion of the fibrillating auricles in man and experimental animals has been visualized and studied. The phenomena seen in the films have been correlated with electrocardiographic and oscillographic records from both man and dogs. Cinematographic observations of auricular fibrillation are reported in the present chapter. Corresponding electrocardiographic and oscilloscopic observations constitute the subject of Chapter XIII

EXPERIMENTAL STUDIES*

Early Investigations: One of the earliest hypotheses concerning the nature of fibrillation was advanced in 1874 by Aubert and Dehn;[23] these authors suggested that the disturbance was produced by injury to a nervous "coordination center" in the heart. Kronecher and Schmey[321] in 1884 produced ventricular fibrillation by puncture of the upper third of the ventricular septum They believed, as did Aubert and Dehn, that the incoordinated beat was due to destruction of a nerve center in the area of the puncture. These views were disposed of by MacWilliam[422] in 1887, who showed that complete spontaneous recovery from fibrillation frequently occurred in the dog. MacWilliam offered the alternative theory that the cause of fibrillation was to be found in the condition of the cardiac muscle itself. He postulated that the excitation wave of fibrillation pursued a sinuous course through the auricles, traveling in a direction determined by the excitability of the muscle fibers in its path. Re-entry of the wave into the original pathway was hypothesized While MacWilliam did not specifically refer to refractory periods and circus movements as such, he clearly anticipated these concepts. MacWilliam's writings, while prophetic in nature, were based upon insufficient experimental evidence to carry conviction and went unheeded by his contemporaries until taken up again in 1918 by Lewis.

Porter[188] in 1894 added the fundamental observation that functional intramuscular blocks in the auricle could deflect the course of the impulse and make it assume devious and redundant paths.

Engleman in 1895,[103] struck by the fact that various parts of the fibrillating auricles were in different phases of contraction at a given instant, postulated the existence of multiple isolated areas of impulse formation throughout the musculature. He stated that ectopic foci discharging simult[illegible]

[illegible] of fibrillation. Engleman's theory of polytopic impulse formation gained the support of leading contemporary

* Much of the following historical material was obtained from the excellent reviews of Lewis[illegible] and Carrey[illegible]

workers in the field, including Lewis and Winterberg, largely because it provided a rational basis for the observation that premature systoles frequently preceded the onset of fibrillation. This theory was later abandoned by Lewis and others in favor of the concept of re-entry.

MacKenzie in 1910 suggested that the auriculo-ventricular node was the cardiac pacemaker in auricular fibrillation, and referred to the disturbance as auriculo-ventricular nodal rhythm.[416]

Rothberger and Winterberg in 1914 conducted the first investigation of auricular flutter and fibrillation[523] by means of direct auricular leads. They rejected Engleman's notion of multiple ectopic impulse formation and proposed the alternative concept of tachysystole, which they described as a tachycardia originating from a single focus at the extremely rapid rate of 3000 to 3500 beats per minute. Their investigations were made on cats in which auricular fibrillation was produced by faradic stimulation. Lewis[385] rejected the concept of tachysystole and felt that the rapid rates described by these investigators were in reality artefacts produced by the electrical stimulation and were not a function of the auricles alone. Garrey's excellent review in 1924 brought forth much additional evidence against the concept of simple tachysystole as the mechanism of auricular fibrillation.[218]

The Circus Movement Theory: The theoretical basis for the circus movement theory of auricular fibrillation was provided by MacWilliam's hypothesis of a re-entry phenomenon together with Porter's intramuscular block experiments.[422] Garrey in 1914 proposed the existence of a circus movement in fibrillation[217] The almost universal acceptance of this theory, however, is a result of the detailed studies of Lewis and his co-workers, instituted in 1918.[148, 362, 369, 373, 381, 384]

As in his investigations of auricular flutter (Chapter V), Lewis' study of the mechanism of auricular fibrillation[369] was based primarily upon an analysis of curves derived from direct auricular lead electrocardiograms of experimentally produced auricular fibrillation. No observations were made on the body of the left auricle. Lewis found it more difficult to establish the existence of a circus movement in fibrillation than in flutter for as he stated: "In measuring records of this type (fibrillation) it is often difficult to fix upon that deflection which most truly represents the passage of the excitation wave. . . . Sometimes the waves pass up the appendix over all contacts; sometimes they pass up and return over the contacts; sometimes in passing up they swerve and meet the contacts at right angles. . . . In attempting to analyze impure flutter, a stage is reached at which, owing on the one hand to the altering form of the auricular complexes, and on the other to the sinuous courses of the centrifugal waves, anything like a complete analysis of the paths taken by the waves becomes impossible."[369]

Nevertheless, from a "broad analysis" Lewis concluded that auricular fibrillation was "a condition in which a *single* excitation wave circulates continuously through the auricular muscle (Figure 230). The path taken both by the central and centrifugal parts of this wave is sinuous and varies in greater or lesser degree from cycle to cycle."[369] In general, the main fibrillation wave was thought to pursue a circular pathway similar to that of the flutter wave except that its course was irregular and sinuous Centrifugal (daughter) waves arising from the main wave were pictured as pursuing an undulant and devious course through the auricular musculature, doubling back upon themselves and spreading to all adjacent muscle fibers that had recovered sufficiently from the previous contraction to be able to contract again.

Thus the results of three years of intense work led Lewis to the conclusion that auricular fibrillation in the experimental animal and, by analogy, in the human subject is due to a wave traveling in a variable and sinuous circus pathway around the caval openings in the auricle.

That Lewis' investigation of fibrillation proved difficult is not surprising when one con-

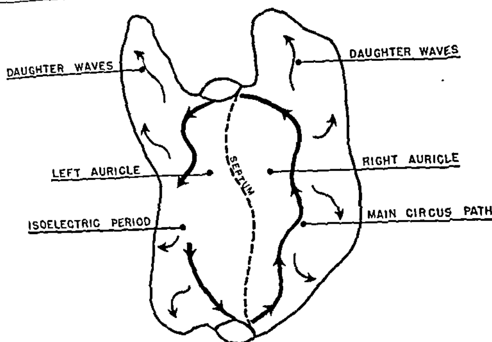


Figure 230 Diagram showing the irregular pathway of the hypothetical circus movement in auricular fibrillation Note the isoelectric gap and the irregular daughter waves

siders the limitations of the equipment available at that time and the inadequate exposure of the heart used in his experiments. Most of Lewis' observations were on auricular flutter; the circus movement theory of fibrillation was derived primarily by analogy with the slower arrhythmia Although the existence of a circus mechanism in auricular fibrillation was suggested originally only as a theory, the concept has taken root and flourished. At present it is accepted more or less as fact in most textbooks covering the subject and by most physiologists, pharmacologists, and cardiologists

Recent Experimental Findings: Experiments performed by a number of observers during the past 30 years have yielded results which appear incompatible with the circus movement theory as proposed by Lewis None of these observers, however, has advanced an alternative concept concerning the mechanism of auricular fibrillation Among the more important recent observations are the following.

Scherf in 1928 attempted to interrupt the main circulating waves of auricular flutter and auricular fibrillation by crushing with a clamp or application of broad ligatures at various sites on the auricles[538] In 16 of 17 experimental animals, the flutter or fibrillation persisted unchanged. Andrus and Carter produced auricular fibrillation in the dog during vagal stimulation by applying a single *induction* shock to the auricular appendix soon after the end of the refractory period. Since no measurable gap appeared on the electrocardiogram between the application of the stimulus and the onset of fibrillation, these authors concluded that the arrhythmia arose at the point of stimulation and not in a ring of muscle at the base of the auricles.[5] Brams and Katz in 1931 reported that complete separation of the right from the left auricle by a deep crush did not alter experimentally produced auricular flutter and fibrillation in the dog as recorded from non-polarizable electrodes on each auricle.[64] Although this finding was considered inconsistent with the circus movement theory as described by Lewis, the

investigators suggested that two new circus movements might have formed, one on either side of the crush.

All experimental studies of auricular fibrillation reported to date have been based upon electrocardiographic interpretation. The high-speed color cinematograph affords an opportunity to clearly visualize the actual events taking place in the auricles during this complex arrhythmia. In the present investigation, cinematographs of experimentally produced auricular fibrillation in over 90 dogs were recorded and subjected to detailed examination. On two occasions a cinematograph of spontaneous auricular fibrillation in a human subject was recorded during auricular appendectomy. The methods of production used and results obtained are described in the remainder of this chapter.

METHODS OF PRODUCTION

In the present series of experiments, four methods were employed to produce auricular fibrillation in dogs: (1) electrical stimulation; (2) application of aconitine; (3) mechanical stroking; and (4) application of acetyl beta methylcholine combined with anoxia.

The auricles in 10 dogs were subjected to rhythmic electrical shocks at a rate above 600 per minute. After stimulation was discontinued fibrillation generally persisted as an after-effect for a few seconds, sometimes for several minutes. In the early experiments auricular fibrillation was studied only during the post-stimulatory phase; motion pictures and electrocardiograms were recorded immediately *after* the stimulation was discontinued. It was soon found that the cinematographic appearance of the fibrillating auricles was identical *during* and *after* stimulation. Hence, in some of the later experiments cinematographic observations were made during both the period of electrical stimulation and the post-stimulatory phase.

The application of aconitine proved most advantageous for our purposes as the arrhythmia was more easily produced and lasted longer than that obtained as an after-effect of electrical stimulation. By mixing the aconitine with India ink the exact point of application could be readily identified on the films and the ectopic focus thus easily located.

Mechanical stroking of the auricle and application of acetyl beta methylcholine combined with anoxia were employed in three or four of the earlier experiments. These methods were soon discarded because the resulting fibrillation was of short duration and difficult to standardize.

Auricular fibrillation in a given animal has been photographed during electrical stimulation, as an after-effect of electrical stimulation, and following aconitine application. The aconitine-induced fibrillation was obtained after sinus rhythm had been re-established following electrical stimulation. *The cinematographic appearance of the auricles in all three instances is identical.* The electrocardiographic patterns recorded immediately after electrical stimulation and following aconitine application also are identical. (Electrocardiographic analysis of the fibrillation obtained during electrical stimulation was impossible because of the numerous deflections caused by the stimuli themselves.) Thus the cinematographic and electrocardiographic appearance of auricular fibrillation is *identical regardless of the method used in its production.*

THE CINEMATOGRAPHIC APPEARANCE OF THE FIBRILLATING AURICLE

When the fibrillating auricle is viewed with the naked eye or on normal speed motion pictures, one sees a shimmering mass in rapid and chaotic motion. No conclusions concerning the nature or mechanism of the disturbance can be drawn from this bewildering spectacle. Because of the rapidity and complexity of the auricular activity, camera speeds up to 3000 frames per second and intense light are necessary to obtain films suitable for detailed study. Even in cinematographs recorded under these conditions, auricular fibrillation appears to con-

sist of a disorganized muscular activity without apparent rhyme or reason, involving all contractile portions of the auricles and differing from any other cardiac state, normal or abnormal, we have previously encountered. This activity has been likened to a pot of boiling water, a storm at sea or choppy waves by various persons who have viewed the films

The cinematographic appearance of auricular fibrillation differs from that of other auricular rhythms in several respects. In normal sinus rhythm as well as in auricular premature systole and auricular tachycardia and flutter, an easily understandable sequence of events is apparent, each contraction wave starts at a designated focus and spreads in all available directions until it is spent, followed by a relaxation wave arising at the same focus and pursuing the same course. No such orderly sequence of events can be detected in auricular fibrillation. In each of the slower rate rhythms, every contraction and relaxation wave starts at the same focus — the wave of normal sinus rhythm at the sino-atrial node, those of auricular premature systole and auricular tachycardia and flutter each at a single ectopic focus — and follows the same course over the auricles

In auricular fibrillation, all movements appear to be independent of the point of origin of the arrhythmia, even when the disturbance is initiated by stimulation at a single site, neither the site of the ectopic focus nor the course of the waves can be determined from the muscular activity The silhouette and surface configuration of all contractile portions of the fibrillating auricles are constantly changing. In normal sinus rhythm, auricular premature systole and most instances of auricular paroxysmal tachycardia, an organized systole of all contractile portions of the auricles is observed Rapid-rate tachycardia and flutter, in which the vigor of the waves varies, may exhibit incomplete systole In auricular fibrillation, although distinct systolic and diastolic changes generally occur, at no time do all contractile portions of the auricles undergo unified systole or diastole

When examined on the high-speed cinematographs (Figure 231), the activity of the fibrillating auricle appears to consist of relatively rhythmic contraction and relaxation waves of variable size traveling in a variable manner over a sea of heterorhythmically contracting, minute muscle segments throughout the contractile auricular musculature. To facilitate description, the motion of the fibrillating auricle has been divided into (1) microscopic or minute activity called "M" activity; and (2) macroscopic or larger activity called "L" activity.

Observation 1: "M" Activity

"M" activity consists of rapid, heterorhythmic contractions and dilatations of minute muscle segments of variable size and shape. Although the individual muscle fibers must undergo periods of systole and diastole, the activity of these minute segments is so rapid and so asynchronous that at no time is a static visual image of the auricle obtained. This seething, tremulous activity is not visible to the naked eye; frequently it gives to the films the appearance of myriads of shimmering, reflected lights. Unfortunately, the effect of "constant motion" is largely lost in the still photographs reproduced here. Although present throughout the contractile portions of the auricles, "M" activity is most clearly seen along the margins of the auricular appendices, especially at the borders of the left appendix which are often serrated with blunt, thin, finger-like projections (Figure 232) The "M" contractions in these marginal areas cause blanching of the tissues; when compared with the dark blue color of contiguous areas in dilatation, the presence of the minute areas of contraction is strikingly demonstrated.

In order to more closely examine the nature of "M" activity, an area of the fibrillating auricle approximately 1 centimeter square was placed under powerful illumination and photographed with special magnifying lens (Figure 233). The intense heat from the strong lights quickly produced secondary arrhythmias, rendering the



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Figure 231. *Stills from* cinematograph of the right auricular appendix of a dog with auricular fibrillation taken a fraction of a second apart with the Fastax camera. Notice the rapid variation in the appearance of the "M" waves. The appearance *of the fibrillating auricle* is constantly changing due to these minute contractions and relaxations.




A




B

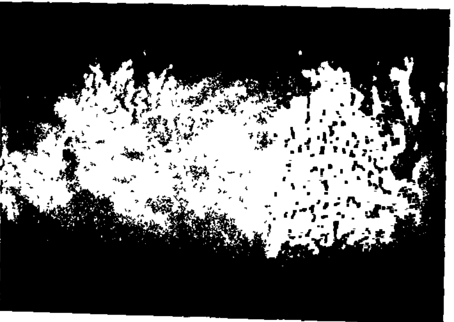



C




D

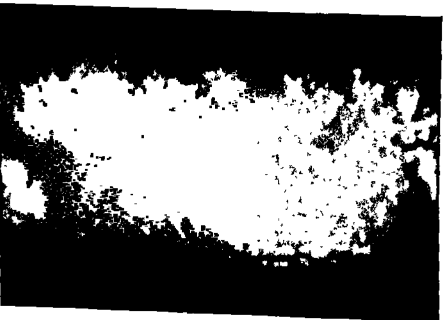
Figure 232. Left auricular appendix in the dog during auricular fibrillation (A), (B) and (C) Auricle in "diastole" produced by an L wave. (D): "Systole" produced by L wave Note the constant change in silhouette of the auricle due to the irregular M activity



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Figure 233. Stills from magnified motion pictures of an area of the left auricular appendix approximately 1 centimeter square. The three pictures were taken a *fraction of a second apart with the Fastax camera. Note* that the appearance of the small contraction and relaxation waves is different in each picture. There is no evidence of a minute circus movement. By projecting these films on a large screen much greater magnification can be *obtained*

auricles unsuitable for study for more than a few seconds; however, as noted in the Appendix, such brief exposure times are adequate for recording high speed cinematographs. When this series of pictures was projected to full screen size, a magnification of several thousand times could be obtained. Contracting muscle segments varying in size from an estimated minimum of 0.2 to 0.8 millimeters in diameter could be identified and their contraction rates fairly accurately timed with a stopwatch. These minute segments were of variable shape and appeared to contract in a completely heterorhythmic manner. At intervals the entire auricular area shown on the screen contracted simultaneous with and apparently independent of the contractions of the minute segments; this phenomenon was attributed to "L" activity. The rate of contraction of the various segments differed greatly, but the majority, regardless of size, contracted at a rate between 800 and 1500 per minute. This experiment was performed 10 times. Fibrillary movements were best seen in the appendices.

Observation 2 "L" Activity

When the fibrillating auricle is viewed on the high-speed cinematographs, contraction and relaxation ("L") waves appear simultaneously with and superimposed on the sea of "M" activity. Although most prominent over the right auricular body, "L" activity is visible in all portions of the auricular musculature except in the noncontractile body of the left auricle. The "L" contractions are relatively rhythmic, occurring at a rate of approximately 400 to 600 per minute even when the auricles are stimulated by electrical shocks at a much more rapid rate. The vigor of "L" waves is extremely variable; unlike "M" contractions, they cover a measurable distance. Both the point of origin and the course of the "L" waves vary during a given bout of fibrillation. The contractions appear to arise from innumerable foci even when the arrhythmia is produced at a single ectopic focus. In the right auricle the majority of the "L" waves travel from right to left (from the caudal toward the cephalic end) even when the stimulus has been applied in the head of the sinus node. Some of the waves, however, proceed in the opposite direction, and occasionally they change from one direction to another during a given bout of fibrillation. This is in contra-distinction to our observations on auricular tachycardia and premature systole, in each of which the contraction waves start at a single ectopic focus and travel away from the focus in all available directions. Not only do "L" waves vary in force and direction but they range from small waves to large ones which appear to involve almost the whole auricle. This variation can be seen by the unaided eye. The distinction between small "L" waves and "M" activity appears to be slight and indeed it may be that the larger waves are fusion phenomena of the minute activity. However, this arbitrary division does in a measure correspond to various electrical variations in auricular fibrillation which are described in the following chapter.

Since the "M" activity is not visible to the unaided eye, the gross appearance of fibrillation is produced by "L" activity. The chaotic picture is probably a result of the rapid, irregular rate combined with the highly irregular force of the "L" waves. However, as noted in Chapters III and V, the auricles in the rapid tachycardias also may present a chaotic appearance due to variations in the force of the contraction waves. Therefore, an attempt to differentiate these arrhythmias by gross visualization, without the aid of slow motion picture films or electrocardiograms, is generally impossible.

Observation 3: "Systole" and "Diastole" of the Fibrillating Auricle

As the "M" contractions of fibrillation are too feeble and too disorganized to propel blood into the ventricles, whatever systolic and diastolic changes the fibrillating auricles undergo are largely a function of the "L" waves. However, the presence of "M" activity modifies the action of the superimposed "L" waves in the following manner: Due to the asynchronicity









Figure 234. Still photographs from motion pictures of auricular fibrillation of the right auricle in the dog. Photographs on left and right developed from same cinema frames by different photographic processes for contrast

(A) The numerous minute (M) contractions are most prominent in the appendix.

(B) Note the large (L) contractions starting at the tip of the appendix M contractions are also present

of the "M" contractions and dilatations, at any *given instant some areas of* the *auricular* musculature are contracted and some are dilated. The constant presence of small relaxed areas interferes with complete auricular systole. Thus, as a direct result of the presence of "M" activity, the magnitude of motion of the fibrillating auricles is diminished and both systole and *diastole are less complete in fibrillation than in* the other auricular arrhythmias. The size of the auricles in fibrillation is generally between the maximal systolic and diastolic sizes of the same auricles during normal sinus rhythm (Figures 232 and 234). The average size of the fibrillating auricles appears to approximate more closely that of the normal organ in diastole *than in systole.*

Despite the interference of the "M" activity, "L" contractions are often of sufficient strength to effect considerable systolic changes in the auricles. On occasion we have observed substantial, well defined "inflow" tracts in the right ventricle produced by the volume of blood expelled *from the right auricle.*

Observation 4. The Onset and Termination of Fibrillation

The transition from flutter to fibrillation, produced by means of aconitine or electrical stimulation, was frequently recorded in the cinema-

tographs. The outstanding changes were the appearance of "M" activity throughout the auricles and an increase in average auricular size.

In several instances we were able to photograph the termination of auricular fibrillation in dogs. The sequence of events was as follows: The "M" activity disappeared suddenly and the "L" waves exhibited a short period of inactivity during which the degree of auricular dilatation, or the diastolic size of the auricle, increased; these changes were followed by the onset of the sinus rhythm or tachycardia. Apparently the disappearance of the "M" activity heralded the termination of the fibrillation

Since "M" activity is present throughout the contractile portions of the fibrillating auricles, including the region of the sino-auricular node, the "M" contractions render the auricles refractory to impulses initiating from the sinus node. Thus, to restore normal sinus rhythm in the fibrillating auricle, measures should be taken which abolish "M" activity (see Chapter XIII).

OBSERVATION 5. CINEMATOGRAPHIC APPEARANCE OF CLINICAL AURICULAR FIBRILLATION

When viewed with the naked eye, auricular fibrillation in the human subject presents a chaotic spectacle similar to that observed in the dog. Few surgical procedures in the chest provide an opportunity to photograph the details of this activity; however, we were able to record cinematographs of the left auricle in a patient with auricular fibrillation during an auricular appendectomy. The subject was a 38-year old white female exhibiting mitral stenosis from old rheumatic heart disease, auricular fibrillation had been present for about 1½ years At least six arterial emboli to the kidneys, head, legs and intestines had arisen from a thrombus in the left auricular appendix The increasing frequency and severity of these embolic episodes despite anti-coagulant therapy necessitated surgical intervention. The left auricle was exposed and the pericardium reflected away from the anterior and left lateral sides; cinematographs at 200 frames per second were then taken before section of the auricular appendix. To the unaided eye the auricle appeared as a glistening, bluish, dilated sac, fimbriated along the anterior margin of the appendix. Except for movement due to irregular ventricular systoles, no muscular activity could be seen. Gentle palpation of the auricular appendix confirmed the diagnosis of auricular thrombosis.

The motion pictures confirmed the observation that no large contraction waves were present in the auricles (Figure 235). However, despite the thrombus and increased intra-auricular pressure, minute muscular activity could be seen clearly. This "M" activity consisted of irregular, chaotic contractions of minute muscle segments throughout all parts of the auricular appendix, particularly along the fimbriated edge. Due to the rapidity and asynchronicity of the contractions, the auricle exhibited a constantly changing silhouette. In all respects the shimmering activity was similar to but not so marked as that observed in the fibrillating auricle of the dog. (No evidence of a circus movement was found.) Increased auricular pressure markedly reduces the visible auricular activity in fibrillation; this factor undoubtedly accounts for much of the decrease in muscular activity seen in this patient as compared with the dog.

These observations indicate that the mechanism of spontaneous auricular fibrillation in the human subject probably is similar to that in the experimental animal. In neither man nor dog has evidence of the existence of a circus movement been found.

CLINICAL CORRELATION AND DISCUSSION

The demonstration of large "L" contractions in auricular fibrillation tends to disprove the traditional concept that the fibrillating auricles are in a state of permanent diastole even though the individual muscle fibers undergo constant fibrillary contractions. On the other hand, our observations are consistent with recent clinical and pathologic findings that fibrillation *per se* is not as important a factor in the production







Figure 235 Left auricular appendix in a patient with mitral stenosis, auricular fibrillation and congestive failure photographed just before the appendix was amputated for multiple emboli. The changing silhouette is similar to that seen in the dog except that the appendix is distended and full of clot. No circus movement is apparent.



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of mural thrombosis or of embolic accidents as was originally thought. In the absence of auricular dilatation or failure, *the strength of the "L" contractions is generally sufficient to prevent significant stagnation of blood in the auricles.* When auricular dilatation occurs as a result of valvular disease or failure from any cause, stagnation occurs and an increased tendency to mural thrombosis develops whether or not the auricles are fibrillating. As noted in Chapter I, even in the presence of normal sinus rhythm, marked dilatation and stagnation of blood in the auricle occurs during auricular failure. *While it is not denied that fibrillation* may increase the tendency to mural thrombosis, we have observed many instances of clinical auricular fibrillation without auricular dilatation or failure in which mural thrombosis or embolic manifestations did not occur. Conversely, many examples of mural thrombosis with embolic manifestations have been seen to occur in the absence of fibrillation; in these patients the auricles were usually dilated or in failure.

Because of the small muscular areas involved, the individual "M" contractions are weak. When the interauricular pressure rises, as in failure, *the "M" contractions disappear. This sequence* of failure with dilatation and disappearance of "M" activity was visualized in the right auricle on cinematographs showing both auricular appendices simultaneously during auricular fibrillation. Throughout the period of failure no "M" activity was seen over the right chamber although it was clearly demonstrated over the relatively well-functioning left appendix. In the absence of "M" activity, the fibrillating right auricle frequently was cinematographically indistinguishable from the auricle in flutter. This explains the observation that occasionally fibrillation appears to occur in only one auricle. An auricle in failure would not appear to be fibrillating; as a result, an erroneous diagnosis of fibrillation in only the other auricle which is *not in failure* might be made.

As discussed in Chapter XIII, the "L" waves probably correspond to the large electrical deflections called "f" waves which, together with other electrical phenomena to be described, *characterize the electrocardiographic pattern* of fibrillation. In auricular fibrillation, however, to an even greater extent than in tachycardia, many auricular deflections occur in the electrocardiogram which are not accompanied by mechanical contractions on the cinematographs. In some oscillographic records we have counted nearly twice as many "f" waves as there were visible "L" waves on the films (see Chapter XIII).

A question which frequently arose during our *cinematographic study of auricular fibrillation* is, "How would a circus movement look in the slow motion picture?" With the realization that any description of such a hypothetical situation must at best be crude, the following answer to this query is offered.

The circus movement as outlined by Lewis consists of two type of waves, a self-perpetuating main or mother wave following in general a circular or oval (elliptical) pathway gives rise to centrifugal or daughter waves which spread throughout the auricular musculature in an extremely variable manner. Theoretically, the main wave pursues a sinuous course through *the fibrillating auricle, traveling* at a constant rate from the inferior vena cava, up the right auricle to the superior vena cava, around which it turns, then, still following a sinuous course, it travels down the left auricle until it again reaches the inferior vena cava and returns to the right auricle. The tissue lying immediately ahead of the main wave must always be in a state of full diastole, ready to receive the oncoming wave.

If waves such as those described above were present in auricular fibrillation, certain characteristics would serve to identify them in high speed cinematographs of the fibrillating auricles. The main wave could be traced with reasonable certainty as the muscle tissue lying in its immediate pathway would be in complete diastole and thus the motionless muscle would present a sharp contrast to the continuous, chaotic motion of the remainder of the fibrillat-

ing auricles When viewed on the slow-motion pictures, the period of diastole not only would be noticeable but would be measurable. Once the main wave is identified, it could be followed along its sinuous course up the right auricle, around the superior vena cava and down the left auricle. The daughter waves arising from the mother wave would appear as contractions of irregular size and force spreading over the main muscle mass of the auricles.

The picture presented by these mechanical events is roughly analogous to a motor-boat speeding over a lake The boat (the main wave) always travels over the lake (auricles) at a constant speed. Its course is sinuous, but in general it follows a circular path The bow of the boat only enters smooth water (muscle in diastole). As the boat speeds along its tortuous course, innumerable waves (daughter waves) of variable size and speed spread from its wake to all parts of the lake in an irregular manner. After completing one circuit the boat *immediately* begins another circuit, the water calms at its approach (iso-electric gap) to permit its passage The boat continues to travel in a general circular path indefinitely but the tortuosity of its course varies greatly.

Careful and repeated study of slow motion cinematographs of experimentally produced auricular fibrillation in over 90 dogs has *failed to* reveal activity even remotely resembling the hypothetical events described above. On the contrary, our observations are incompatible with the existence of circus movement for the following reasons

(1) When both auricles are viewed simultaneously, they are seen to contract *in one* of three manners: (a) simultaneously, (b) either one slightly before the other, and (c) either one many times more often than the other If circus movements were present, one auricle should always contract before the other; when measured on the high speed cinematographs, the time interval between the contractions of the two auricles would equal several seconds. Furthermore, the number of overall contractions of the two auricles should be identical

(2) Many of the large ("L") waves seen on the cinematographs are broad, often involving the entire width of the auricle from the auriculoventricular groove to the intercaval region. They do not turn to embrace the venae cavae as would occur if they pursued a circus pathway. No centrifugal daughter waves are seen to spread from the main wave. (The presence of such daughter waves was believed to account for the irregular ventricular response characteristic of auricular fibrillation. An alternative explanation for this irregular response will be offered in Chapter XIV.

(3) An essential assumption of the circus movement theory is the constant presence of an excitable gap on the circus pathway; this gap must be in full diastole ready to accept and transmit the oncoming main wave. Such a concept is incompatible with the observation that minute ("M") contractions and dilatations are continually present throughout the contractile portions of the fibrillating auricles.

(4) The fact that *no* circus movement can be seen on high speed cinematographs of either or both auricles does not preclude the existence of minute circus movements occurring in areas of the auricles too small to be demonstrated without further magnification. The presence of such minute circus movements would be visible in the cinematographs recorded under high magnification which are described earlier in this chapter. In these as in all other cinematographs examined in this study, no circus movement was seen.

SUMMARY AND CONCLUSION

Experimentally produced auricular fibrillation in over 90 dogs has been visualized in high speed cinematographs of each auricle, of both auricles simultaneously, and of an area of the auricle approximately 1 cm. square photographed under high magnification. Auricular fibrillation is a chaotic disturbance characterized by complex, continuous, rapid motion different from that observed in any other cardiac state The activity in the fibrillating auricle ranges from asynchronous contractions and di-

latations of minute (microscopic) muscle segments to broad contraction and relaxation waves involving large areas of the contractile portion of the auricles. At a given instant both extreme as well as all intermediate types of contractions may occur simultaneously with and superimposed on one another.

For purposes of elucidation, the motion of the fibrillating auricles has been divided into (1) microscopic or "M" activity; and (2) macroscopic or "L" activity.

(1) High speed cinematographs recorded under high magnification demonstrate the existence of rapidly contracting muscle segments throughout the contractile portions of the auricles. Due to the heterorhythmic nature of this microscopic activity, some areas of the auricles are in contraction while others are in dilatation. "M" activity is diagnostic of auricular fibrillation; no contractions of similar appearance have been observed in any other auricular arrhythmia.

(2) "L" activity consists of contraction and relaxation waves of variable vigor which occur throughout the contractile auricular musculature simultaneous with and superimposed on the "M" contractions and dilatations. "L" activity is responsible for the gross appearance of auricular fibrillation. The "L" waves differ from the excitation waves of the other rhythms in that they arise from diverse foci *regardless of the site of production of the arrhythmia* and change their course during a given bout of fibrillation.

The vigor of the "L" waves may be sufficient to produce well-defined systolic and diastolic changes in the auricles. The simultaneous presence of minute contractions and dilatations ("M" activity) throughout the contractile musculature, however, prevents complete systole and diastole. The size of the fibrillating auricles is between the maximal systolic and diastolic sizes of the normal organ, nearer the normal diastolic size.

Cinematographs of the fibrillating left auricle of a patient with mitral stenosis were recorded during auricular appendectomy. These films reveal minute, chaotic, heterorhythmic muscular contractions similar to the "M" activity seen in the dog.

No iso-electric gap, no centrifugal or daughter waves, and no circus movement exist in the fibrillating auricle.

CHAPTER XIII

The Electrical Activity of the Fibrillating Auricles

Previous investigations of the electrical activity of the fibrillating auricles have been limited by the relative insensitivity of the electrocardiographs available. As Lewis acknowledged, the complex nature of fibrillation could not be fully elucidated with the equipment used in his studies 30 years ago. Despite these limitations, the circus movement theory of auricular fibrillation achieved general acceptance and comparatively little new evidence concerning the mechanism of this disturbance has been advanced since the theory was introduced 30 years ago.

The cinematographically recorded experiments described in the previous chapter demonstrate that fibrillation in the dog *consists of* multiple, chaotic, rapid and asynchronous contractions of varying size and force occurring simultaneously throughout the contractile portions of the auricles. These cinematographic observations require a complete re-evaluation of the electrical activity of the fibrillating auricles in both man and animals. Part I of the present chapter concerns the electrical counterparts of the mechanical events of fibrillation in the dog, Part II considers the arrhythmia as it occurs in man

Part I

AURICULAR FIBRILLATION IN DOGS

EQUIPMENT AND METHODS

A preliminary series of experiments was recorded by the direct-writing electrocardiograph Experiments were performed in over 50 dogs In 15, auricular fibrillation was produced by faradic stimulation and studied during the post-stimulatory phase, in the remaining 35, the arrhythmia was produced by local application of aconitine to the wall of the auricle. Records were made from the standard limb leads and from non-polarizable electrodes placed directly on the auricles. In each instance the results duplicated the tracings of Lewis and other workers. In the limb leads, the base line was irregular and showed only the familiar "f" waves of fibrillation (Figure 236A). In direct auricular leads, large deflections with frequencies as high as 1800 to 2000 impulses per minute were inscribed (Figure 236B). These rates approximated the maximum which the equipment was capable of recording accurately. Even at the maximum paper speed of 50 millimeters per second the deflections were crowded into such a narrow space that adequate analysis of the tracings was impossible. Records obtained with the photographic recording Sanborn Twin-Beam Electrocardiograph (Appendix) (Figures 237 and 238) revealed considerably greater detail than did the direct writing machines. Nevertheless, the limitations of both these instruments were such that they were incapable of accurately recording the electrical events in the fibrillating auricles. They were therefore abandoned in favor of more sensitive apparatus.

The cathode-ray oscillograph, an inertia-free instrument with a wide frequency response, was the chief tool used in our electrographic studies of auricular fibrillation. Following preliminary studies with various types of relatively crude oscillographic equipment, the Du Mont 279 dual-beam cathode-ray oscillograph and the various types of recording equipment described in the Appendix were utilized. Initially, a

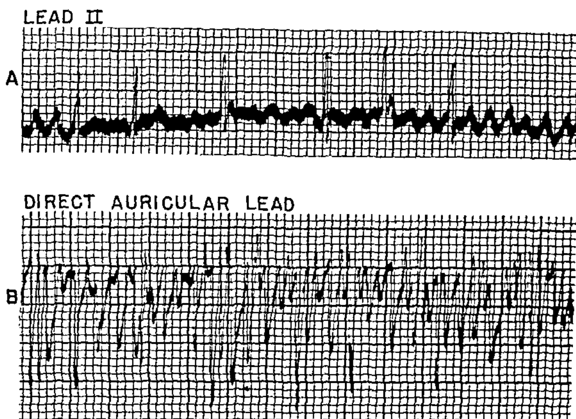


Figure 236. Electrocardiograms of auricular fibrillation recorded on standard direct writing instrument (enlarged two times).
(A) [illegible]
(B) [illegible] es the limited frequency response and mech [illegible] the equipment is inadequate and does not record all of the electrical activity of the disturbance.

power-line operated four-stage electronic pre-amplifier was used with this instrument; later, two battery-operated Grass P 4 pre-amplifiers were substituted. Records were made from standard and unipolar limb leads and from unipolar direct auricular leads. Soft-tipped nonpolarizable copper-copper sulfate electrodes (Appendix), held manually, were generally employed.

Auricular fibrillation was produced by aconitine application in each of 35 animals As described in the Appendix, low concentrations (0.05 to 0.5 per cent) of the alkaloid were used initially, necessitating repeated application during which accidental contamination of the ventricle frequently occurred with resultant ventricular fibrillation. Later in the study a 5.0 per cent solution of aconitine was found more suitable. This solution could be applied to a restricted focus not more than 2 or 3 millimeters in diameter; auricular fibrillation usually appeared within 30 seconds and was of relatively long duration.

THE OSCILLOGRAM OF AURICULAR FIBRILLATION IN THE DOG

Tracings obtained with each of the oscillographs, pre-amplifiers, and methods of recording described above appeared identical

Each type of complex recorded from the right auricle was present in tracings from either the body or the appendix of the left auricle.

Observation 1: *Single Unipolar Direct Auricular Leads*

Oscillograms of auricular fibrillation obtained from single unipolar direct auricular leads consistently showed irregular, bizarre, chaotic complexes of astonishingly high frequency. The rapidly moving fluorescent dot seen on the oscillograph tube front appeared to the naked eye as "chain lightning." No isoelectric period (au-

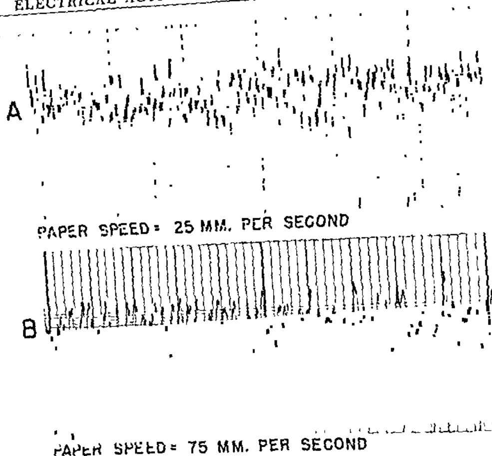


Figure 237 [illegible] cular leads recorded by Sanborn Twin- [illegible] e more rapid rate there is such crowd-

ricular diastole) was inscribed (Figure 239) The deflections included a wide range of sizes and frequencies For descriptive purposes, the oscillographic deflections of auricular fibrillation have been divided into (1) minute; and (2) large complexes

Minute Complexes: These deflections are of extremely small potential, estimated to range between 50 to 500 microvolts. They are seen most clearly at those periods when the large waves formed "plateaus"; here the small waves are visible as sharp spikes or gentle waves along the crest of the plateau (Figures 240 and 241). The minute complexes also appear as slurring of the up or down stroke of the large deflections. Because they are "buried" in large waves or are present on the plateaus, their frequency could not be calculated exactly. Whenever they occur in bursts and can be counted, the frequency varies approximately from 7,000 to 40,000 impulses per minute. The rate is not constant or cyclic, but is irregular and bizarre. The configuration of the minute deflections changes constantly, the majority appear as spikes and the remainder as small, rounded waves The electrical events responsible for these minute deflections are too weak and too rapid to be recorded by standard electrocardiographic equipment.

Large Complexes: These deflections of constantly changing configuration, now spiked and peaked, again broad and slurred, occur at irregular rates of 500 to 1,000 per minute (Figures 242 and 243). Their potential is estimated at roughly 3 to 10 millivolts, often equal to that of a normal P wave. The large complexes

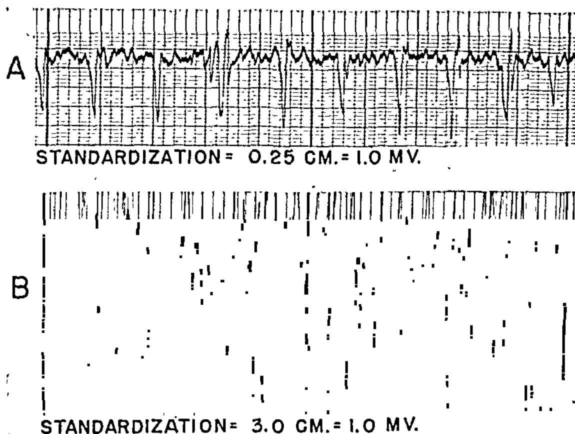


Figure 238 Auricular fibrillation in a dog. Direct auricular leads recorded by Twin-Beam Electrocardiograph to demonstrate effect of increasing standardization on configuration of electrical activity of the arrhythmia Oscillations which are not present in A appear in B





Figure 239. Direct auricular lead cathode-ray oscillograms from a dog.

(Upper) Regular sinus rhythm. Note the smooth base-line.

(Lower) After the production of auricular fibrillation, the electrical activity has changed completely.

Each mark in upper portion of each oscillogram represents 1/120 second.

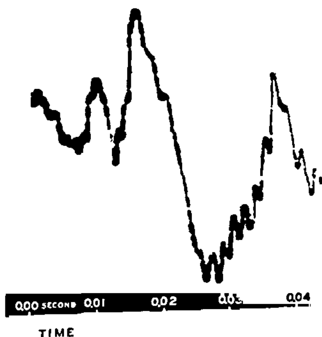


Figure 240 Direct auricular lead cathode-ray oscillogram of auricular fibrillation in a dog The burst of minute activity between times 0 03 and 0 04 are at a rate of approximately 42,000 impulses per minute

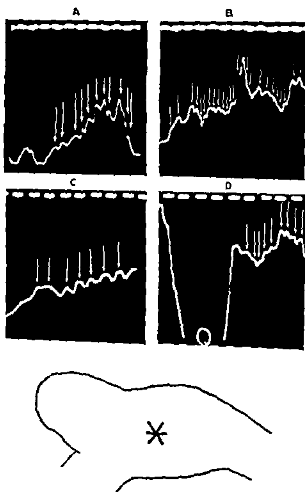


Figure 241. Direct auricular lead cathode-ray oscillograms from 4 dogs with auricular fibrillation. Each mark in the upper part of each oscillogram represents 1/120 second. The large number of small waves of various types (arrows) have frequencies up to 26,000 per minute. (D) illustrates the relative potential of this activity as contrasted to a ventricular complex (Q).

Reduced ⅓ full oscillographic tube-front size

were recorded from all areas of both auricles. They undoubtedly correspond to the familiar "f" waves of the electrocardiogram.

An intermediate type of complex frequently is seen in leads taken from the appendices. These deflections resemble the large complexes but appear smaller in potential, exhibit greater uniformity in configuration, and occur with somewhat greater regularity and higher frequency (Figure 244). The frequency of the intermediate complexes varies from an estimated 1,200 to 3,000 per minute, their potential is estimated at 0.5 to 3 millivolts. These deflections are just within the range of the standard electrocardiograph; they probably correspond to the "grass-like" base line and the irregularities of the "f" waves seen in limb lead electrocardiograms of auricular fibrillation.

Observation 2: Two Simultaneous Unipolar Direct Auricular Leads from the Right Auricle

In order to further explore the nature of the electrical activity in the fibrillating auricles, in

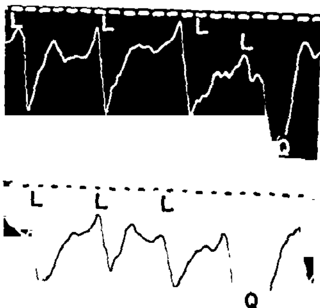


Figure 242. Direct auricular lead cathode-ray oscillograms from two dogs with auricular fibrillation. These figures illustrate types of large (L) activity. The waves often appear regular in frequency, but close examination shows they are actually irregular. Their configurations are always different.

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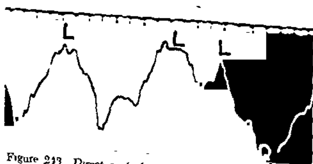


Figure 243. Direct auricular lead cathode-ray oscillogram during auricular fibrillation in the dog. This demonstrates constantly changing configuration of the large (L) waves.

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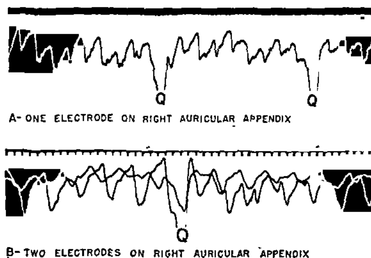


Figure 244. Direct auricular lead cathode-ray oscillograms during auricular fibrillation in the dog.

(A) Intermediate type of activity recorded from a single electrode.

(B) Two electrodes ¼ inch apart on right auricular appendix There is almost complete dissimilarity of the activity recorded from the two electrodes.

Reduced ⅔ tube-front size.

each of 35 experiments two unipolar direct auricular leads were recorded simultaneously on the dual-beam cathode-ray oscillograph. The following combinations of positions of the electrodes were used: (1) One electrode was placed at the aconitine focus, the other at one of various distal points throughout the body and appendix of the right auricles (Figure 245). (2) Two electrodes were placed at varying distances apart at numerous selected sites over the right auricular body and appendix (Figure 246). (3) One electrode was maintained in a fixed position at a given site; the other was moved in a circle around the fixed electrode at a radius of 0.5 to 1.0 centimeters (Figure 247).

Minute and large complexes were present throughout each record from any one of the leads described above. A comparison of the tracings recorded simultaneously from any of the three combinations of electrode positions revealed a general lack of synchronicity between records; when synchronism was present, it was usually of brief duration Characteristically, the deflection in one lead first preceded, then followed that in the other simultaneous lead. At no time was a consistent pattern or sequence apparent. The frequencies and potentials of each type of deflection were different in each lead. In a few pairs of simultaneously recorded leads, usually from the appendix, the large complexes exhibited some degree of uniformity; however, these complexes were never identical. This lack of correlation was observed with each of the three combinations of electrode positions utilized (Figures 245, 246 and 247).

Observation 3: The Onset and Termination of Auricular Fibrillation

During numerous experiments, transitions occurred between tachycardia and fibrillation. The change recorded by the oscillograph in these instances was a dramatic one. The smooth baseline and regularly repeated complexes of auricular flutter were replaced by the "chain lightning" appearance of fibrillation (Figure 248). The transition from fibrillation to a slower rhythm was equally abrupt. This transition was observed spontaneously, following freezing of the focus, or following the use of antifibrillary drugs (Chapter XVI).

Observation 4: Semi-Direct Auricular Lead Oscillograms

In five experiments, records were made from electrodes placed on extracardiac mediastinal structures (esophagus, superior and inferior

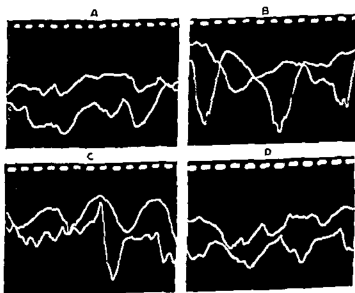


Figure 245 Direct auricular lead cathode-ray oscillograms of auricular fibrillation in a dog High magnification of electrical activity, one electrode (top tracing) on aconitine focus, the other at various points on the auricle

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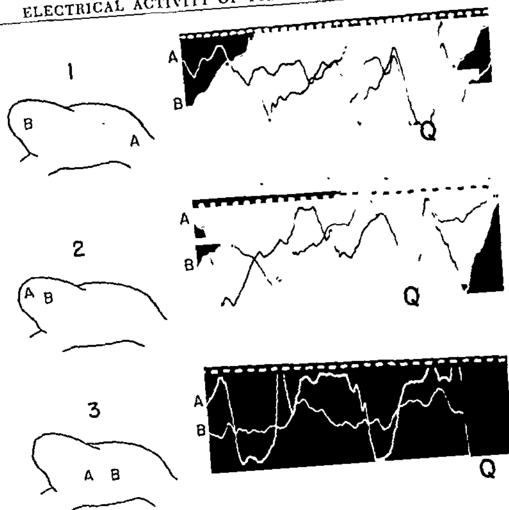


Figure 246 Direct auricular lead cathode-ray oscillograms fro… icle in a dog.
(1) …
(2)
(3)
Note
Redu

venae cavae, pulmonary artery and veins and lungs). In each instance there was a consistent change from the electrical pattern obtained from direct leads; this change was consistent with that expected according to the inverse-square law. When the electrodes were placed at distances as small as 0 5 centimeters from the auricle, the minute waves virtually disappeared from the tracings The potential of the larger waves became smaller and their configuration less jagged. As the distance between the electrode and the auricle increased, the potential progressively decreased until, in oscillograms recorded several centimeters from the auricle, the pattern resembled that ordinarily obtained from indirect limb or chest leads.

DISCUSSION

The non-polarizable copper-co… sulfate ele… approxi- … As dem- …ated in the magnified motion pictures of the fibrillating auricle (Chapter XII), the electrode tip was in direct contact with numerous muscle fibers undergoing chaotic contractions. Any electrical events associated with the motion of these muscle bundles would be recorded by the electrode. In addition, those impulses from

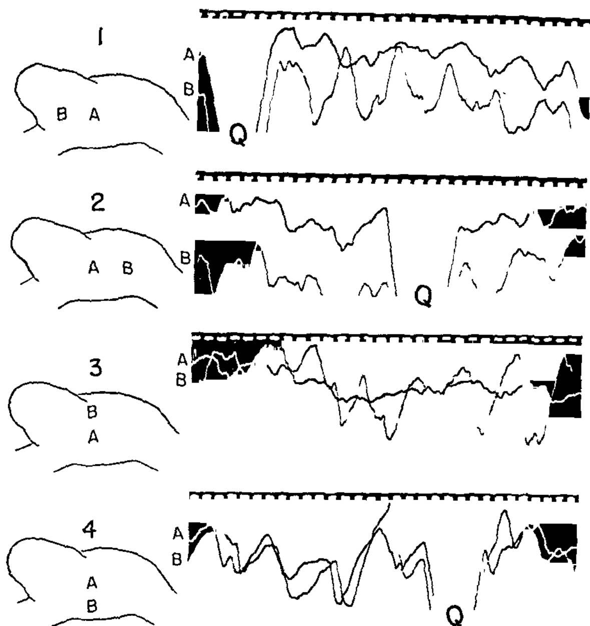


Figure 247. Direct auricular lead cathode-ray oscillograms from a dog with auricular fibrillation. In these four experiments electrode A was kept at a fixed point. Electrode B was moved around electrode A as shown in the diagram to the left There is complete dissimilarity of electrical activity recorded. Reduced ⅔ tube-front size

distant, noncontiguous portions of the auricle which were productive of sufficient potential to be transmitted to the electrode also would be recorded. Thus, the deflections in the oscillograms must represent electrical activity in muscle fibers directly beneath the electrode and a summation of all impulses from other portions of the auricle powerful enough to reach the area beneath the electrode.

Dissociation between mechanical and electrical events in the auricle has been observed frequently during our studies of the arrhythmias. This phenomenon is exemplified as the rate of discharge from an ectopic focus increases during tachycardia In such instances the frequency and potential of the electrocardiographic deflections are unchanged but many mechanical contractions become progressively weaker until eventually some of them drop out completely. Consequently, fewer waves are seen on the cinematographs than can be accounted for by the number of *deflections inscribed* on the electrocardiogram. The principle of *dissociation between mechanical* and electri-

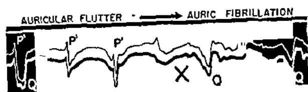


Figure 248 Simultaneous direct auricular lead cathode-ray oscillograms in dog Transition from flutter to fibrillation is marked X The activities of each rhythm are completely different
Reduced ⅔ tube-front size.

cal events apparently applies also to auricular fibrillation Despite the rapidity of motion in the fibrillating auricle, the number of mechanical contractions occurring in a small area of the auricle photographed under high magnification is but a small fraction of the number of electrical impulses recorded from the same area during the same period. Both cinematographically and electrically, the waves of fibrillation are much more rapid and more complex than those of any other arrhythmia This discrepancy between the rates of mechanical and electrical activity is greatest when the auricle is distended or the heart is in failure, here only the "stronger" ("L") waves are seen on the cinematographs while the electrocardiographic pattern exhibits both the minute and the large complexes of fibrillation.

The orders of electrical activity — large, intermediate, and minute — for the most part appear to correspond to the large ("L"), intermediate and small ("M") activity distinguished in high speed cinematographs of the fibrillating auricle in the dog

Large Complexes: The large ("L") waves of fibrillation seen on the cinematographs constitute the only motion of the auricles of sufficient magnitude to correspond to the electrical activity reflected in the large waves of the oscillograms. Both occur at approximately the same rate The large waves on the oscillograms vary in amplitude and configuration during a given bout of fibrillation As noted in Chapter XII, the large contraction waves of auricular fibrillation differ from the waves of any other disturbance in that not only may they change direction during their course over the auricle, but apparently they originate at diverse areas. It thus appears reasonable to assume a correlation between the alteration in potential and configuration of the large deflections on the oscillograms and the change in direction of propagation and focus of initiation of the contraction waves as observed cinematographically.

Minute Complexes: As noted in Chapter XII, one of the unique cinematographic characteristics of auricular fibrillation is the presence of numerous minute contractions and relaxations ("M" activity) throughout the contractile portions of the auricles. The minute deflections seen on the oscillograms are present only in fibrillation, thereby distinguishing it electrographically from all other auricular rhythms. The implication that the minute deflections are an electrical counterpart of the minute contractions is further strengthened by the high frequency and low amplitude of each. Only these minute deflections are of sufficiently rapid rate and small size to account for the minute contractions on the cinematographs. The minute electrical waves are of such low amplitude that they are not recorded in tracings from electrodes even a short distance from the auricle. Therefore, the majority must arise in the area beneath the electrode. The principle of dissociation between mechanical and electrical activity is well demonstrated in "M" mechanical and electrical activity of auricular fibrillation.

Certain fundamental differences distinguish the electrical activity of auricular fibrillation from that occurring in normal sinus rhythm, auricular premature systoles, paroxysmal tachycardia and auricular flutter. In each of the slower-rate rhythms, a P or P' wave is present. Except for the occurrence of P or P', QRS, T and U waves, the baseline is smooth and straight. Furthermore, simultaneous leads from any combination of electrode positions always show definite synchronism and relationship of electrical events; that is, differences in the configuration of the auricular deflections and/or the time of onset of the intrinsic deflections in the tracings are correlated with differences in location of the recording electrodes with respect to the focus.

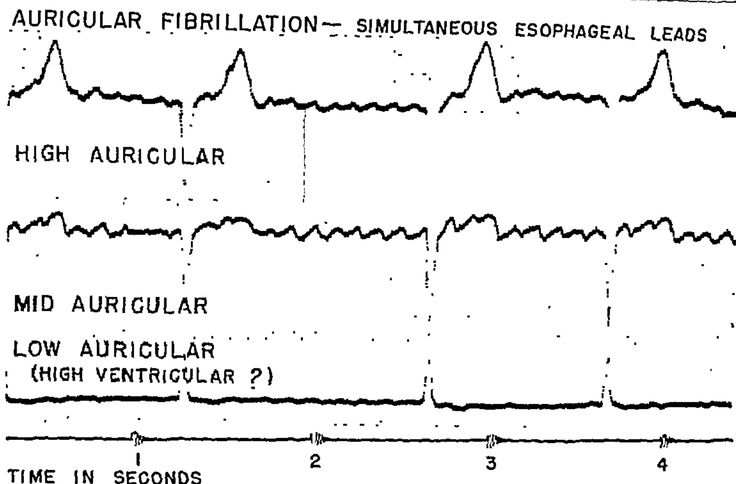


Figure 249. Three simultaneous esophageal leads from different auricular levels recorded at maximum amplitude and at double speed. The tracings show typical pattern of auricular fibrillation. No synchronicity between the auricular deflections can be seen

It is impossible to demonstrate the direction of the excitation waves or the presence of a circus movement.

In contrast with all other auricular rhythms, both normal and abnormal, fibrillation never exhibits a P or P′ wave. Except for ventricular deflections, the baseline is *always* completely irregular and broken, showing the deflections described above. *Finally, except for the ventricular complexes, simultaneous leads from any* combination of electrode positions generally display complete asynchronism and little apparent relationship of one lead to another.

As stated earlier, the circus movement theory of fibrillation has been widely accepted despite Lewis' recognition of the inadequacy of supporting evidence. This theory has been questioned only rarely in recent years. The type of electrical activity manifested in oscillograms from various parts of the fibrillating auricles of the dog is chaotic and bizarre Furthermore, in no tracing was evidence of an isoelectric period recorded. Lewis considered that ". . . the presence of some gap is all-essential, upon its existence the continued movement absolutely depends."[369] Therefore, even if a circus movement could be conceived in the presence of the constant, turbulent electrical activity found in the fibrillating auricle, the specific factor which Lewis held essential to his theory has not been demonstrated.

One modification of Lewis' circus movement theory predicates that the mother wave pursuing the circus pathway is microscopic in size. Careful examination of oscillograms inscribed from small electrodes in a large number of experiments confirms the visual evidence obtained by cinematographs taken with magnifying lenses: Neither the tracings nor the films reveal any evidence of a circus movement, either *macroscopic or microscopic*, in the fibrillating auricle of the dog.

In summary, our cinematographic and elec-

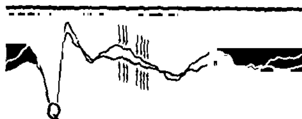


Figure 250 Simultaneous esophageal lead cathode-ray oscillograms from a patient with auricular fibrillation Somatic tremor is easily identified as its potential is small and it affects both electrodes equally (arrows).
Reduced ⅔ tube-front size.

trographic studies of experimentally produced auricular fibrillation in the dog are mutually confirmatory. Auricular fibrillation appears to consist of chaotic, bizarre, mechanical and electrical activity of a type heretofore undescribed.

Part II

AURICULAR FIBRILLATION IN MAN

Little or nothing is known concerning the mechanism of auricular fibrillation in man. While gross arrhythmia of the pulse beat has been recognized for many decades, not until 1899 was a correlation between the experimental and clinical disturbances suggested by Cushny[122] Lewis first proposed the circus movement theory as an explanation of experimentally produced fibrillation in dogs[360] Later, Lewis, Drury and Iliescu studied the arrhythmia in man and described the large deflections now known as the "f" waves of auricular fibrillation, they calculated from these waves that impulses travel in a roughly circular pathway through the fibrillating auricles.[373, 375] Since Lewis' investigations, little has been added to our knowledge of the activity which takes place in the fibrillating auricle of man Brown[74] and Nyboer and Hamilton[470] recorded esophageal lead electro[illegible] an effort t[illegible] ance, the [illegible] electrocardiographic equipment used in their studies revealed only a rough undulatory baseline similar to that seen in more conventional leads We confirmed the findings of these and other workers by recording three simultaneous esophageal leads with the standard electrocardiograph (Figure 249). Definite conclusions concerning the nature of auricular fibrillation could not be established from such records.

EQUIPMENT AND METHODS

As in our study of auricular fibrillation in dogs, the dual-beam cathode-ray oscillograph proved superior to the standard electrocardiograph as a means of exploring the nature of electrical activity in the fibrillating auricle of man. In the present investigation, multiple esophageal and limb lead oscillograms were recorded in each of 24 patients with auricular fibrillation. Eleven tracings were recorded from four undigitalized subjects; the remaining 20 subjects were fully digitalized.

Before describing the results of these studies, four artefacts which were encountered on occasion must be considered. (1) The most important artefact found was somatic tremor. In some subjects, especially those in whom repeated

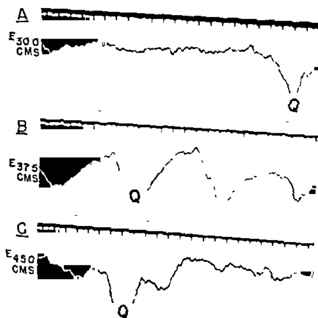


Figure 251. Thre[illegible] from a patient [illegible] The greatest acti[illegible] supra-auricular r[illegible] [illegible] 450 cms is over the ventricles
Reduced ⅔ tube-front size

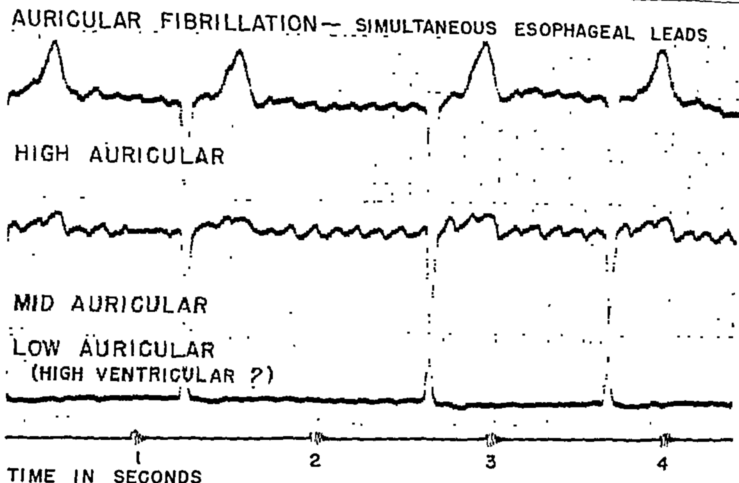


Figure 249. Three simultaneous esophageal leads from different auricular levels recorded at maximum amplitude and at double speed The tracings show typical pattern of auricular fibrillation. No synchronicity between the auricular deflections can be seen

It is impossible to demonstrate the direction of the excitation waves or the presence of a circus movement

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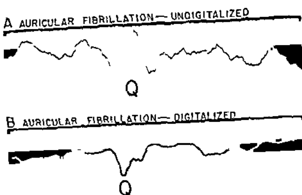


Figure 254 Esophageal lead cathode-ray oscillograms from a single patient Recorded at different speeds.
(A) Undigitalized.
(B) Fully digitalized Note the pronounced effect of digitalis on the electrical activity of the fibrillating auricle

esophageal lead oscillogram of fibrillation in man was identical with the records obtained from semi-direct auricular leads in dogs.

Simultaneous Unipolar Esophageal Leads: Simultaneous oscillograms were recorded from two electrodes placed 2.5 to 5 centimeters apart in the esophagus directly behind the auricles In every instance the tracing from each electrode exhibited the small and large waves described above. The two fluorescent dots from the electron beams traveled in constantly varying directions, crossed back and forth over each other and inscribed waves of different amplitude and configuration (Figure 253). Rarely, for brief intervals, the waves in the simultaneously recorded tracings were inscribed synchronously. As in the animal, however, careful examination revealed that the complexes in the two tracings were not identical.

The large waves in the simultaneous esophageal lead oscillograms of fibrillation in man appeared more synchronous than those in simultaneous direct auricular leads in the experimental animal This was explained by the fact that a certain number of the waves had sufficient potential to be picked up by both esophageal electrodes with the result that they were inscribed almost synchronously.

Similar observations were made in multiple simultaneous esophageal lead electrocardiograms (Figure 249). Since the electrocardiograph has lower magnification and lower frequency response than the oscillograph, only the largest waves ("f" waves) were recorded with fidelity. These large deflections were so irregular and asynchronous that they seemed incompatible with the presence of a strong impulse traveling from one end of the auricle to the other in a definite pathway. An attempt to correlate the electrical waves with a definite course over the auricles, as was done in our study of flutter, proved futile. Some waves appeared to travel upward through the auricle, others downward; some were found at only one level.

Supra-Auricular and Ventricular Levels: Oscillograms from these levels consistently resembled those from various limb leads (Figures 251A and C). The large waves appeared as gentle undulations and no spikes were inscribed. On closer examination, minute, almost microscopic, irregularities were found superimposed on each wave. Deflections of similar configuration were obtained in oscillograms from leads directly behind the auricles when the records were made at low amplification.

OBSERVATION 6. THE EFFECT OF DIGITALIS

As noted earlier, 20 of the 24 patients studied were completely digitalized. The oscillograms of the digitalized patients were much less irregular than records from undigitalized subjects (Figure 254). The effect of digitalis on auricular fibrillation is discussed in greater detail in Chapter XVI.

DISCUSSION

Except for changes attributable to digitalis, results were similar in all patients examined by esophageal lead techniques. Records at repeated intervals in each patient were similar. Several were well trained subjects; one was studied on six occasions, another on three, others twice each. One patient initially exhibited auricular flutter which later was converted to fibrillation (Figure 252). The records obtained at fast recording speeds (25.4 centimeters per second) during bouts of flutter were

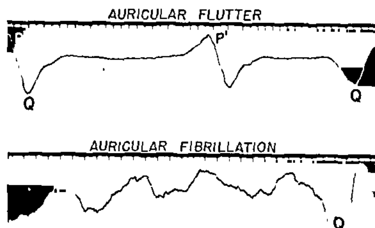


Figure 252. Esophageal lead cathode-ray oscillograms from a single patient.

(*Upper*) During auricular flutter. The base line is smooth at all times.

(*Lower*) *After conversion to auricular fibrillation.* Activity characteristic of the arrhythmia is now present

Reduced ⅔ tube-front size.

records were made, this factor was negligible; *in others, it could not be eliminated. The pres*ence of somatic tremor was easily recognized because the bizarre, irregular deflections produced by the tremors were parallel and identical in each of two simultaneous esophageal leads (Figure 250). Regardless of how well trained and calm the individual subjects were, it was impossible to record chest or limb leads *on the cathode-ray oscillograph without picking* up tremors from underlying striated muscle. The artefact was minimal in unipolar esophageal leads because *no striated* muscle was present between electrode and heart. Thus, esophageal leads provided the only method of investigating the electrical events of fibrillation in man. (2) Alternating current artefacts were *avoided by use of a wire-screen cage to shield* the subject (Appendix) (3) Respiratory movements which could cause a shifting baseline in the esophageal electrocardiogram were controlled by instructing the subject to hold his breath at the midpoint of the respiratory cycle. (4) Transmitted aortic or ventricular pulsations which might produce a large wave immediately *following the ventricular T wave* were eliminated by moving the electrode a few millimeters up or down the esophagus.

THE ESOPHAGEAL LEAD OSCILLOGRAM OF AURICULAR FIBRILLATION IN MAN

Observation 5: Esophageal Leads from Undigitalized Patients

Auricular Levels: Cathode-ray oscillograms were made from esophageal levels directly posterior to the auricles. The records were remarkably similar to the direct auricular lead oscillograms obtained in animals. No P or P′ wave was present. The baseline was irregular and jagged (Figure 251), in contradistinction *to the smooth and regular baseline characteris*tic of normal sinus rhythm and the slower-rate auricular arrhythmias (Figure 252). As in the animal, the *oscillogram of fibrillation in man* exhibited both small and large waves. (1) Small, irregular waves, usually *0.1 millivolt* or less in amplitude, occurred at frequencies as high as several thousand per minute. (2) Large waves, approximately *0.2 to 1.0 millivolt in* amplitude, appeared at frequencies of approximately 350 to 600 per minute; these large waves *were readily recognized as the familiar "f"* waves of fibrillation. In fibrillation in dogs, oscillograms recorded from the esophagus or other mediastinal structures differed from the direct auricular lead oscillograms in that, even at *short distances from the auricles, the ampli*tude and frequency of the minute waves tended to diminish and the large waves were of slower *frequency, smaller amplitude,* and were more rolling and less peaked in configuration. The



Figure 253 Simultaneous esophageal lead cathode-ray oscillogram from a patient with auricular fibrillation The leads are 2 5 cm apart. Note dissimilarity of pattern.

Reduced ⅔ tube-front size

direct leads generally are not apparent in semi-direct leads, but probably manifest themselves as slight irregularities in the large waves.

As fibrillation is converted to auricular flutter either spontaneously, by freezing the aconitine focus or by the use of antifibrillatory drugs, the chaotic waves disappear and are replaced by a smooth baseline and regularly repeated complexes. When flutter is converted to fibrillation, the converse occurs.

Auricular fibrillation in 20 digitalized and four undigitalized patients was studied by means of multiple esophageal leads recorded on the cathode-ray oscillograph. These tracings were similar with semi-direct auricular lead oscillograms recorded in dogs with auricular fibrillation before and after treatment with digitalis. Large waves similar to those seen in direct lead tracings from dogs were recorded in the esophageal oscillograms from man. The tracings from esophageal leads exhibited fewer small complexes than those from direct leads, presumably because the minute electrical activity was too weak to be transmitted through the esophageal tissues to the electrodes. Tracings from two simultaneously recording electrodes placed 2.5 to 5 centimeters apart in the esophagus were generally asynchronous and unsymmetrical. Only the largest waves were synchronous, since these waves were of sufficient voltage to be transmitted to both electrodes.

The electrical events in the fibrillating auricles are identical in man and in the dog. Both cinematographically and oscillographically, auricular fibrillation is a bizarre, chaotic disturbance characterized by constant, asynchronous activity unlike that observed in any other auricular arrhythmia.

characteristic of that disturbance; regular auricular deflections, smooth and flat baseline, synchronism between tracings from multiple simultaneously recorded leads were present. During fibrillation, all the characteristics of flutter were replaced by the typical jagged appearance of the tracings from the other 23 patients. The electrographic differences between flutter and fibrillation were clearly demonstrated in this patient.

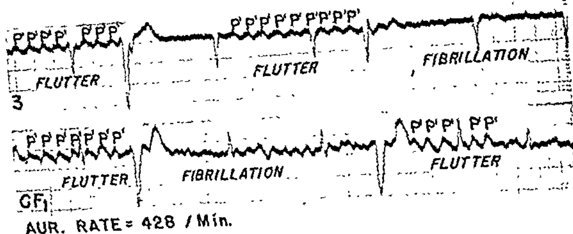
The electrical activity observed in man during auricular fibrillation closely parallels that found in the experimental animal. This evidence concerning the identity of the electrical events in the fibrillating auricles of man and animals would appear to be the most conclusive yet obtained. In man, as in the animal, the activity is continuous, bizarre and chaotic. In esophageal leads from man, to an even greater extent than in the direct leads from dogs, the electrodes record activity occurring in auricular areas much larger than that in direct contact with the electrode. Waves of large potential undoubtedly are recorded from a distance. The fact that no isoelectric interval is apparent in oscillograms of auricular fibrillation does not indicate that each muscle fiber is continuously active; as shown in magnified cinematographs, the individual muscle bundles undergo a period of relaxation following each contraction. But as one bundle enters "diastole," its neighbor, also recorded by the oscillograph, contracts. These asynchronous contractions are responsible for the continuous, irregular undulations described The oscillogram of auricular fibrillation in man in no way indicates that the electrical wave follows an irregular circus pathway suggestive of a circus movement, nor do the periods of "diastole" recorded from semi-direct esophageal or indirect limb leads represent the isoelectric gap which Lewis considered essential to the existence of a circus movement. These oscillographic observations on spontaneous auricular fibrillation in man are completely analogous to our cinematographic and electrographic observations of the experimentally produced arrhythmia in dogs.

SUMMARY AND CONCLUSION

The electrical activity of the fibrillating auricles in man and in the dog was studied by means or the cathode-ray oscillograph, the direct-writing electrocardiograph, and the photographic recording twin-beam electrocardiograph. Because of almost unlimited amplification and frequency response as well as freedom from mechanical inertia, the cathode-ray oscillograph used with a high film-speed recording apparatus proved the most useful of the various instruments available.

Auricular fibrillation in the dog was produced by mechanical, electrical and aconitine stimulation, the electrically initiated arrhythmia was studied during the post-stimulatory phase. Identical oscillograms were recorded regardless of the method of production employed. Direct lead oscillograms from the fibrillating auricles of the dog reveal the presence of continuous, bizarre, chaotic and non-cyclic electrical activity which, for descriptive purposes, may be divided into two types: (1) minute waves with an amplitude of 50 to 500 microvolts and a frequency of 7,000 to 40,000 per minute; and (2) large waves 3 to 10 millivolts in amplitude, recorded at a rate of 400 to 1,000 per minute. The large waves in the oscillogram correspond to the familiar "f" waves of the fibrillation electrocardiogram. Intermediate types of waves are frequently inscribed; these vary in amplitude from 0.5 to 3 millivolts and in frequency from 1,200 to 3,000 per minute. Two simultaneously recorded direct leads show only rare synchronism whether the electrodes are placed close together or far apart.

Oscillograms from electrodes placed in mediastinal structures adjacent to the auricles, such as the esophagus or venae cavae, exhibit deflections of substantially lower frequency and potential than those recorded directly from the auricles In tracings recorded even a short distance from the auricle, the large complexes are smaller and smoothed out; these closely resemble the "f" waves of the fibrillation electrocardiogram. The small waves recorded from



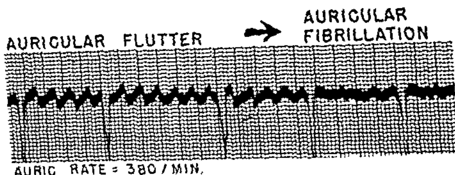


Figure 255 Electrocardiograms from two patients with auricular flutter at rates near the fibrillation threshold (A) The auricular rate is 428. (B) The auricular rate is 380

shown in Figure 256B, conduction of the impulse also is slowed when marked shortening of the cardiac cycle occurs as a result of rapid beating

Observation 1 Cinematographic Appearance of the Auricles During the Establishment of Auricular Fibrillation

The effects of accelerating the auricular rate in experimental animals have been studied extensively by means of the high speed cinematograph Before the fibrillation threshold rate is reached, contractions are seen to arise from the ectopic focus from whence they spread outward in wave-like fashion across the entire surface of the auricle. As the auricular rate approaches the fibrillation threshold, the contraction waves travel at progressively *slower* rates; the onset of each contraction wave occurs sooner after the termination of its predecessor, and auricular diastole is thereby shortened. Despite the progressive decrease in the speed of the contraction waves and the length of diastole, the auricles continue to undergo coordinated and orderly contractions as the auricular rate approaches the fibrillation threshold. When the threshold is reached suddenly the auricles fail to respond to the stimuli in an orderly manner and no cordinated systole or diastole is seen; instead, the chaotic motion of auricular fibrillation is present (Chapter XII).

By timing the duration of diastole in high-

CHAPTER XIV

Further Considerations on the Nature of Auricular Fibrillation

THE PRODUCTION OF AURICULAR FIBRILLATION

AURICULAR RATE

IT HAS LONG been known that experimental fibrillation is produced when the auricular rate exceeds a certain critical level. This has been termed the fibrillation threshold rate. In the dog anesthetized with pentobarbital or morphine and urethane, a rate from 400 to 650 must usually be achieved before fibrillation appears. Although considerable variation exists from dog to dog, in the individual animal under uniform circumstances the fibrillation threshold rate appears to be fairly constant

In five humans it was possible to determine the threshold rate necessary for the establishment of auricular fibrillation. Electrocardiograms from these patients exhibited frequent transitions between auricular flutter and fibrillation (Figure 255). In such instances the auricular rates immediately prior to the establishment of fibrillation approximate the fibrillation threshold. These rates were 360, 380, 428, 430 and 432 per minute respectively. The fact that all five patients had heart disease and some were receiving digitalis undoubtedly influenced the fibrillation threshold. It is of interest that the threshold rate in these patients is not radically different from that observed in the experimental animal

INTRA-AURICULAR CONDUCTION

Cinematographic studies described in preceding chapters reveal that the contraction wave is conducted across the auricles at a greatly reduced speed when premature auricular systoles or very rapid auricular rates are induced by either electrical stimulation or aconitine. Lewis, Feil and Stroud [365] originally made similar observations regarding the excitation waves of auricular premature systoles and the rapid-rate arrhythmias. By stimulating the auricle electrically at one point and recording the arrival of the excitation wave (onset of the intrinsic deflection) at another point, these investigators demonstrated that as the rate of stimulation was increased, the rate of conduction of the excitation wave decreased. Slight widening and notching of the P′ wave occurred when conduction was thus slowed by rapid stimulation. Love [394] noted that if stimulation of the auricles is sufficiently premature, excitation takes place with little or no conduction of the excitatory process. This observation indicates that conduction recovery takes place after the absolute refractory period has ended. Instances in which the excitation wave travels for short distances with a gradual and premature termination have been termed decrement conduction Similarly, we have observed cinematographically that the contraction waves of certain auricular premature systoles travel only a short distance over the surface of the auricle before dying out (Chapter II).

The foregoing observations support the conclusion that following excitation, the auricles gradually recover their ability to conduct the next excitation and contraction wave. This is illustrated by the theoretical auricular conduction recovery curve in Figure 256A. Stimulation applied to the auricle at time-point X in the cardiac cycle would result in a premature systole which would be poorly conducted. As

Figure 257 Direct lead from the left auricle of a dog during rhythmic stimulation of the right auricular appendix (tracing recorded at double speed and enlarged 2 X.)

ute ([illegible]

ute [illegible] that the stimulus-intrinsic intervals are longer at the more rapid rate In tracing (B) there is a tendency toward alternation in the length of the intervals Changes in the width of the P' wave are more clearly seen in the simultaneously recorded lead from the right auricle shown in Figure 258

speed cinematographs recorded while the rate of stimulation of the auricles is gradually increased, the effect of auricular rate on the length of auricular diastole has been determined. As shown in Figure 150, the shortening of the cardiac cycle which occurs at extremely rapid auricular rates is primarily at the expense of the diastolic period. Thus the "rest period" of auricular muscle grows progressively shorter as the auricular rate nears the fibrillation threshold. In auricular fibrillation there is no unified diastole of the entire auricle, although diastole of individual muscle fibers is seen to occur.

The electrical counterpart of the above observation was encountered in our study of auricular conduction recovery.

Figure 258 Direct lead from right auricle recorded simultaneously with tracing in Figure 257

(A) During stimulation at a rate of 222 (Arrows denote stimuli)

(B) During stimulation at a rate of 600 The P' waves are slightly wider at the faster rate The stimulus-intrinsic interval also is increased, because the recording electrode is closer to the stimulating electrode, changes in the stimulus-intrinsic intervals are not as marked as those in the lead from the left auricle (Figure 257).

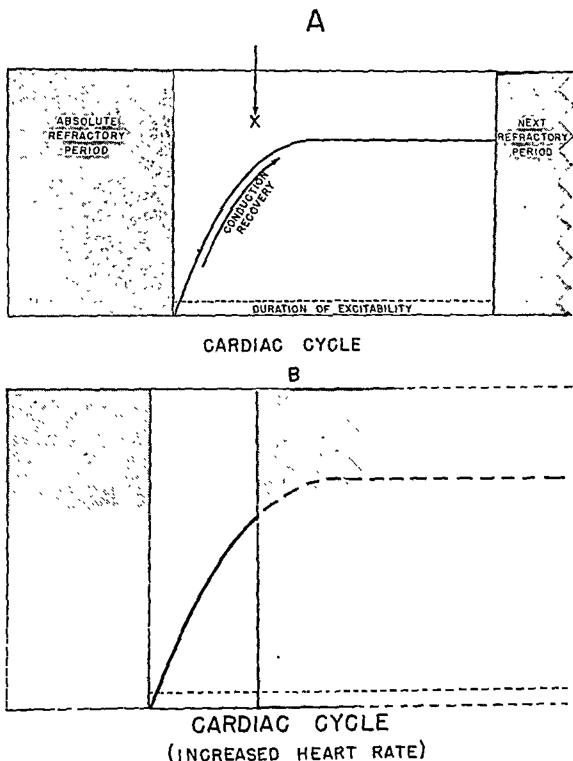


Figure 256 Hypothetical figure illustrating conduction recovery

(A) Conduction recovery taking place after the absolute refractory period has ended Extrasystole occurring at point (X) would be conducted poorly because excitation takes place before conduction recovery is completed

(B) Drastic shortening of the cardiac cycle occurring at rapid auricular rates If the auricular rate is extremely rapid, conduction recovery is incomplete and conduction is slowed This conduction slowing is similar to that produced by premature excitation but differs quantitatively

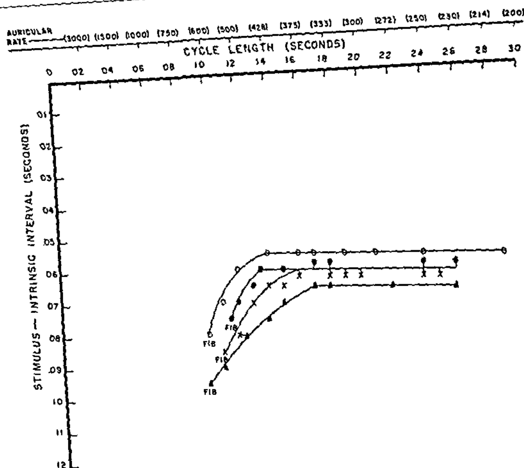


Figure 259 Auricular conduction recovery. Ordinates (Y axis) shows stimulus-intrinsic interval (transmission interval) in seconds as measured in direct lead electrocardiogram from left auricle recorded while the right auricle was being electrically stimulated Abscissae (X axis) shows the stimulus-stimulus interval (cycle interval) in seconds For reference the corresponding auricular rate is shown above the stimulus-stimulus interval

The figure shows the normal auricular conduction recovery curves for four dogs. No effort was made to obtain identical placement and recording of stimulating electrodes in the four experiments, however, once the recording and stimulating electrodes had been positioned they were left unchanged for the duration of the experiment As shown by the curves, the auricular conduction remains normal over a wide range of rates When the rate is increased to levels above 400, however, auricular conduction fails rapidly and, in the experiments illustrated, leads to the appearance of fibrillation The lowest point on each curve represents the fibrillation threshold. (Urethane anesthesia – Dogs BO, BK, JF, LF.)

failure At pre-fibrillatory auricular rates, splintering and slight widening of the P' wave occurs as conduction begins to fail This pattern was termed impure flutter by Lewis.[366] Since such aberration of the P' wave occurs after conduction has become impaired, it is conceivable that a further impairment of conduction would produce additional splintering and breakdown of the auricular deflection, culminating in the chaotic oscillations characteristic of fibrillation

Observation 3. The Effect of Altered Auricular Conductivity on the Fibrillation Threshold

Digitalis and quinidine are known to decrease the *conductivity* of the auricles. Vagal stimulation and acetylcholine on the other hand have been shown under certain circumstances to increase the conductivity of the auricles.[152, 376, 493] The effect of these agents on conduction recovery and the fibrillation threshold was studied by associating auricular conductivity

Observation 2: Auricular Conduction Recovery

Conduction recovery of the heart can be studied by two methods: (1) measuring the speed of conduction of extrasystoles produced at various intervals throughout a controlled cardiac cycle; and (2) measuring the conduction in the auricle at various cardiac rates. The second method was employed as follows:

In 17 dogs, the auricles were exposed in the manner described in the Appendix. A stimulating electrode from a low frequency pulse generator was attached to the right auricular appendix, and recording electrodes were placed on the body of the right auricle and on the left auricular appendix. The positions of all electrodes remained constant throughout the experiment. The auricle was stimulated at rates in gradual increments of 25 to 50 stimuli per minute while continuous electrocardiograms were recorded at double speed (50 millimeters per second). In a few experiments, direct auricular leads were recorded on the cathode-ray oscilloscope at a camera speed of 7½ inches per second. By measuring the stimulus-intrinsic intervals* in the continuous tracings, changes in conduction associated with the accelerating auricular rate could be determined (Figure 256) Since the stimulus-intrinsic interval is longer in the leads from the left auricle, this lead was chosen for measurement. This interval includes the latent period as a negligible and constant error.

As observed in cinematographic studies, the threshold rate for the production of auricular fibrillation in the dog lay around 600 per minute.** As the rate of stimulation nears the critical threshold rate (about 600 impulses per minute) two events become apparent: (1) the stimulus-intrinsic interval begins to increase (Figure 257); and (2) the P′ waves become wider and slightly splintered (Figure 258). These changes rapidly become more marked as the auricular rate is further increased. When the threshold rate is reached and fibrillation is produced, the stimulus-intrinsic interval can no longer be measured. In some animals the fibrillatory pattern appears *during* the gradual acceleration of the rate of stimulation but conduction improves and flutter recurs when the rate of stimulation is again constant. With a further slight increase in the rate, a degree of conduction failure is produced from which the auricles cannot recover and fibrillation persists as long as the high rate of stimulation is maintained. Occasionally, auricular fibrillation persists for a short time after the electrical stimulation is discontinued.

These events leading to the production of fibrillation are summarized in the auricular conduction recovery curves shown in Figure 259. These curves have been constructed by plotting the stimulus-intrinsic interval against the stimulus-stimulus interval (rate of stimulation).* It is apparent that fibrillation occurs after conduction has begun to fail.

The cinematographic and electrocardiographic evidence presented in Observations 1 and 2 indicate that auricular fibrillation occurs when the auricular rate is accelerated to a point beyond which the auricles no longer conduct and contract in an orderly manner This evidence further suggests that the fibrillatory state represents an advanced degree of conduction

* A constant point on the stimulus signal was chosen and the interval between this point and the onset of the intrinsic deflection was measured

** In approximately half of the animals studied, we were unable to obtain sufficiently rapid auricular rates for the production of fibrillation It is not completely clear why certain auricles respond to extremely rapid rates of stimulation while others do not Observations suggest that this protective mechanism may be related to the gradient of conduction recovery. Other observations of this question are presented in Chapter XVI.

* Curves of similar shape can be constructed by plotting the width (duration) of the P′ wave against the P′-P′ interval (auricular rate) but the intricacy of making such measurements renders the latter method of little practical value for the auricles Such is not the case for the ventricles Decherd and Ruskin[136] have constructed ventricular conduction recovery curves by plotting the width (duration) of the QRS complex against the R-R interval (ventricular rate) Lewis and Masters[57] and Ruskin and Decherd[135] constructed recovery curves for auriculo-ventricular conduction by plotting the P-R interval against the P-P or preceding R-P interval It is of considerable interest that such curves have a similar shape whether they represent auricular, ventricular or A-V conduction

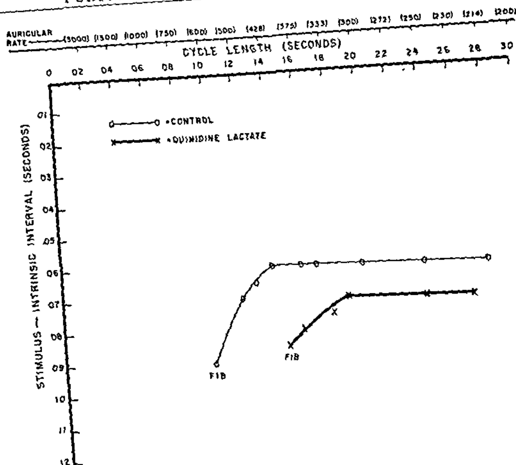


Figure 261 Auricular conduction recovery before and after administration of quinidine In the unmedicated animal auricular fibrillation appeared when the auricular rate reached 600 per minute Following the administration of quinidine (8 mgm./Kg.) a state of decreased conductivity prevailed and auricular fibrillation appeared when the auricular rate reached 400. (Urethane anesthesia – Dog AS.)

chemical reactions leading to the formation and discharge of an impulse in heart muscle. Until the nature of the normal sinus impulse is better understood, the precise mechanisms and reactions responsible for the development of an ectopic focus necessarily remain obscure Nevertheless, certain factors which apparently contribute to the development of auricular fibrillation can be enumerated. These factors must either (1) favor the establishment of the rapidly discharging ectopic focus present in fibrillation, (2) increase the rate of discharge from a pre-existing ectopic focus to beyond the fibrillation threshold, or (3) impair conductivity to a point at which the fibrillation threshold drops to below the rate of discharge from a pre-existing ectopic focus.

Vagal Tone: The effect of increased vagal tone on the production of auricular fibrillation has long been recognized In the experimental animal auricular fibrillation has been observed to result from simple vagal stimulation[371] The administration of a cholinergic drug to patients with hyperthyroidism may precipitate the arrhythmia.[404] Fibrillation following the administration of toxic doses of digitalis may be due in part to increased vagal tone. Acetylcholine or vagal stimulation frequently leads to the discharge of volleys of rapid impulses in isolated auricular muscle.[53, 317] A single stimulus applied to the auricle shortly after the refractory period has been found to produce auricular fibrillation only if the vagus is stimulated simultaneously.[5] In a preceding paragraph the effect

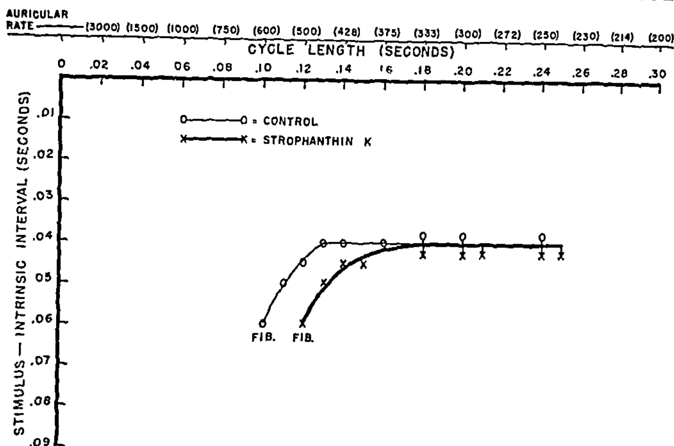


Figure 260 Auricular conduction recovery before and after administration of strophanthin Auricular fibrillation in unmedicated animal appeared when the auricular rate reached 600 per minute After administration of strophanthin K (0 030 mgm/Kg) fibrillation appeared when the auricular rate reached 480 per minute (Urethane anesthesia — Dog EC)

with auricular rate. As shown in Figure 260 before strophanthin, an auricular rate of 600 was required for the production of fibrillation, whereas, following digitalization an auricular rate of 480 resulted in fibrillation A similar result was obtained following the administration of a small dose of quinidine to another animal. As shown in Figure 261, under control conditions, an auricular rate of 600 resulted in fibrillation, whereas, following quinidine administration an auricular rate of 400 resulted in fibrillation With acetylcholine, the opposite effect was observed (Figure 262): Under control conditions, an auricular rate of 590 produced fibrillation, whereas, in the same animal during administration of acetylcholine fibrillation was not produced until the auriclular rate reached 1500. *These observations clearly show that two variables exist for the establishment of auricular fibrillation: (1) auricular rate and (2) auricular conductivity.* Although Lewis and associates [371] made similar observations with regard to the effect of vagal stimulation on the fibrillation threshold, they held the view that the vagus was incapable of accelerating fiber conduction. However, as shown in Figure 262 acetylcholine accelerated conduction recovery, and between the auricular rates of 360 and 1200 resulted in a super-normal phase of conduction recovery.*

From these observations the conclusion is drawn that *conduction failure must take place before auricular fibrillation can exist.* Our studies further suggest that the degree of conduction failure necessary for the establishment of fibrillation may be fairly constant.

FACTORS PREDISPOSING TO THE PRODUCTION OF AURICULAR FIBRILLATION

Little is known concerning the electro-bio-

* This phenomenon has been observed in all animals studied but the degree and the auricular rate at which it occurs varies somewhat in different animals (Figure 303, Chapter XVI) Similar acceleration of intra-auricular conduction at rapid rates of stimulation has been noted by Rosenbleuth and Ramos.[491]

of the vagus substance (acetylcholine) on auricular conductivity was considered. Clinically and in the experimental animal, the effect of the vagus on auricular rate appears to overshadow its effect on conductivity. Vagal stimulation during auricular flutter often causes an increase in auricular rate sufficient to produce a transition to fibrillation (Figures 194, 195). This and other events resulting from an increase in vagal tone are discussed in Chapter XVI.

Auricular Distention: Stretching of auricular tissue has been found by Scherf, Scharf and Goklen[547] to produce auricular fibrillation. We have confirmed this observation by inserting a balloon into the auricle through a purse string opening so that the stem of the balloon remained outside the heart. When the auricle was distended by injecting saline into the balloon, auricular fibrillation frequently developed (Figure 263).

Anoxia: Apparently anoxia has a complex effect on the heart, tending to depress conductivity and increase vagal tone.[441] We have observed that anoxia frequently converts aconitine-induced flutter to fibrillation (Figure 264), the rhythm quickly reverts to flutter when anoxia is relieved. The presence of anoxia has been found to favor the production of auricular fibrillation by cholinergic drugs[465] and may be a factor in post-operative fibrillation. The fact that auricular fibrillation is uncommon in patients with anoxia resulting from severe emphysema or one of the cyanotic varieties of congenital heart disease may be attributable to the chronicity of the anoxia in such instances.

←
Figure 262 (A) Auricular conduct...
rates ... per minute ... auricular
Dog MP) ... (Urethane anesthesia —
(B) Portion of the electrocardiogram from the same animal during administration of acetylcholine exhibiting electrically produced auricular flutter at a rate of approximately 900 per minute. Arrows denote stimuli (Recorded at double speed and enlarged two times.)

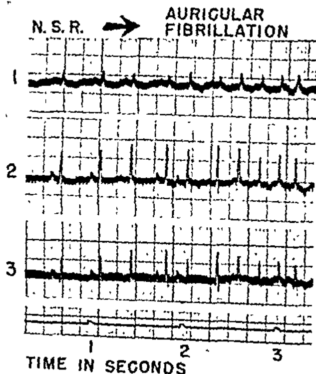
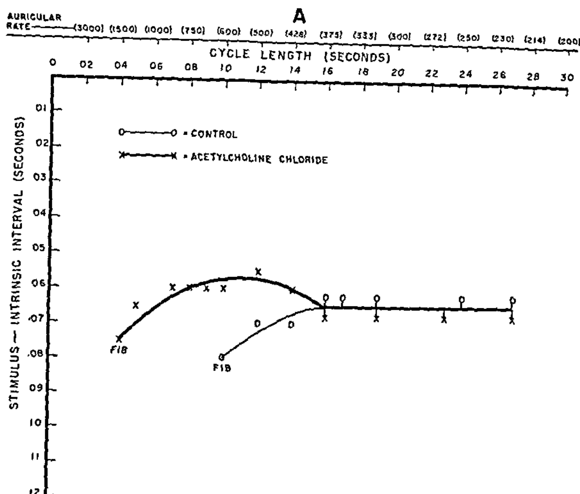


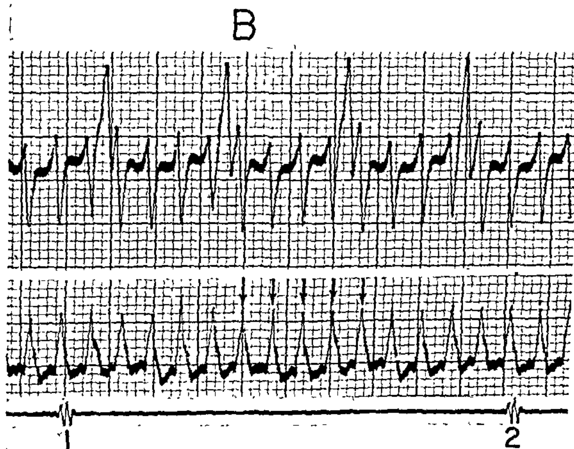
Figure 263. A rubber balloon was placed in the right auricle of a dog during normal sinus rhythm. As the balloon was inflated it distended the auricle. Note the onset of auricular fibrillation with the auricular distention.

Heart Failure: Heart failure is well known to favor the production of auricular fibrillation. This may be attributable to the presence of both auricular distention and anoxia. Auricular fibrillation is a frequent complication in mitral stenosis which is accompanied by marked left auricular distention, even in the absence of clinical heart failure.

Myocardial Infarction: This condition is frequently complicated by auricular fibrillation and other arrhythmias. The existence of heart failure or anoxia may be contributing factors. In two of our patients with auricular fibrillation complicating myocardial infarction fresh auricular infarct... cardi... ...ctro- the f... ...of the arrhythmia was in ... that junctional areas (Figure 316).

Age: Auricular fibrillation is rare in children under the age of 1 year, infrequent in children under the age of 12 years and progressively more frequent during and after middle age until

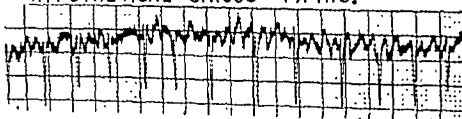

A
AURICULAR RATE
(3000) (1500) (1000) (750) (600) (500) (428) (375) (333) (300) (272) (250) (230) (214) (200)
CYCLE LENGTH (SECONDS)
0 02 04 06 08 10 12 14 16 18 20 22 24 26 28 30
STIMULUS — INTRINSIC INTERVAL (SECONDS)
01 02 03 04 05 06 07 08 09 10 11 12
o——o = CONTROL
x——x = ACETYLCHOLINE CHLORIDE
FIB
FIB


B
1
2

A.



B. BEFORE BURN TO INTERRUPT HYPOTHETICAL CIRCUS PATHS.



AFTER BURN FIBRILLATION CONTINUES AS BEFORE.

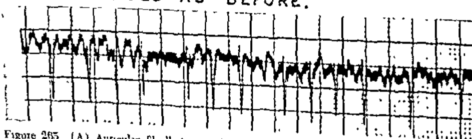


Figure 265 (A) Auricular fibrillation in the dog showing the outline of an inverted "T" burn which transects Lewis's hypothetical circus pathway Fibrillation was initiated from a focus on the appendix before the burn was inflicted

(B) Electrocardiograms recorded before and after the burn are identical.

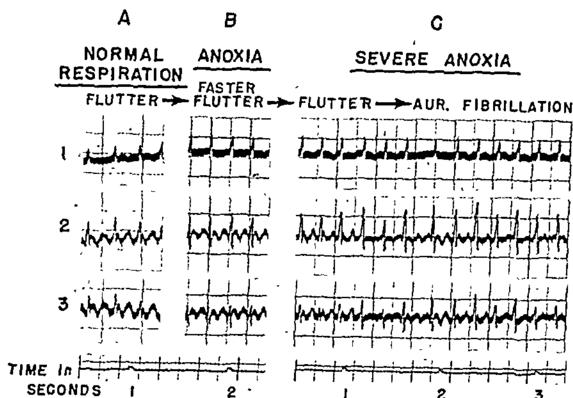


Figure 264 Electrocardiogram showing the effect of anoxia on flutter
(A)
(B)
(C)

in senility it is by far the most common arrhythmia. An analysis of the age distribution of a series of 329 patients with auricular arrhythmias examined at the Los Angeles County General Hospital is shown in Table II.

TABLE II

AGE INCIDENCE OF AURICULAR FIBRILLATION IN RELATIONSHIP TO OTHER AURICULAR ARRHYTHMIAS *

Table	*Total*	*Under 60 years*	*Over 60 years*
Auricular fibrillation	220	25%	75%
Auricular flutter	85	31%	69%
Paroxysmal auricular tachycardia	24	71%	29%

* Statistics compiled by Mrs R S Cosby of Los Angeles

The relative rarity of auricular fibrillation among young individuals may be due partially to the high degree of myocardial conductivity in infancy; as noted earlier, this factor would tend to raise the fibrillation threshold On the other hand, the greater incidence of fibrillation, especially of the paroxysmal type, among aged persons may reflect the increase in the frequency of other forms of heart disease in older age groups.

Idiopathic Auricular Fibrillation: In an estimated 4 to 6 per cent of all patients with auricular fibrillation the etiologic factors are unknown. Idiopathic auricular fibrillation has occasionally been encountered in several members of the same family. In these instances none of the factors ordinarily associated with auricular fibrillation could be found.

THE PERPETUATION OF AURICULAR FIBRILLATION

The mechanism by which auricular fibrillation is perpetuated has been the subject of much investigation and speculation. Present controversies center chiefly around the question of whether fibrillation is perpetuated by one rapidly discharging focus, multiple foci, or a self-sustaining re-entrant phenomenon, termed the circus movement.

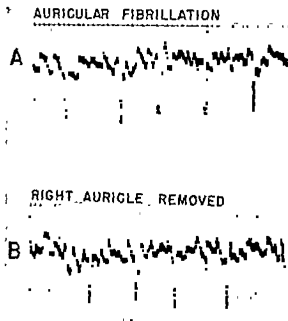


Figure 267 (A) Auricular fibrillation from an aconitine focus on the left auricle

(B) The right auricle was removed following production of the arrhythmia Electrocardiograms recorded before and after the operation (resection) are identical

nitine to the caudal end of the right auricle. Continuous limb leads and direct auricular leads from the right and left appendix were taken The entire right auricle except the ectopic focus on the posterior surface was frozen with ethyl-chloride spray until the area was completely covered with white frost After this procedure, the right auricle appeared motionless and the direct lead tracings indicated that all electrical activity in the right chamber had ceased while the left auricle continued to fibrillate. In one instance all visible portions of both auricles with the exception of the focus on the posterior surface of the right chamber were frozen, the limb lead electrocardiograms continued to record fibrillation, probably due to electrical activity in the interauricular septum and area surrounding the focus (Figure 266).

Observation 7 Complete Removal of One Auricle

Auricular fibrillation was produced by local application of aconitine to the left auricular appendix. Limb lead electrocardiograms and direct leads from the appendix of the left auricle were recorded continuously. While fibrillation was present, the venae cavae were clamped and the entire right auricle was removed by a series of rapid incisions along its attachment to the ventricle. This procedure was completed in less than 20 seconds. As seen in Figure 267, auricular fibrillation persisted in the left auricle after the venae cavae and right auricle had been completely separated from the heart.

In summary, observations 4, 5, 6 and 7 are consistent with the results of blocking experiments reported by other investigators and with cinematographic and oscillographic observations described in preceding chapters. These data when considered together conclusively prove that auricular fibrillation is not perpetuated by a circus or re-entrant phenomenon.

UNIFOCAL vs. MULTIFOCAL THEORY OF AURICULAR FIBRILLATION

In this and in studies by many other investigators fibrillation has been repeatedly initiated by electrical stimulation or aconitine application at a single focus in the auricles. Whether the original point of stimulation remains the sole site of impulse formation or whether multiple other sites subsequently become established as ectopic foci discharging impulses at independent rates is not yet determined.

The multifocal theory of auricular fibrillation is based primarily upon electrocardiographic observations. When direct or semi-direct leads from the fibrillating auricle are recorded on standard electrocardiographic equipment, only the larger ("L") complexes can be seen (Chapter XIII). Because these large deflections are variable in configuration and occur at irregular intervals (Figure 249), the hypothesis has been advanced that they represent impulses arising at multiple ectopic foci discharging at irregular rates. If each of the large waves is initiated by the discharge of an impulse from an independent site of stimulus formation, there would indeed be foci in all regions of the auricles. However, cinematographic observations reported in Chapter XII indicate that the ir-

THE CIRCUS MOVEMENT THEORY

As noted in Chapter XII, Scherf, Brams and Katz have attempted to interrupt the electrical activity of the fibrillating auricle by blocking the hypothetical circus path with a broad ligature or a deep crush; these procedures failed to terminate the arrhythmia. It was concluded that such results, although incompatible with the circus movement theory as advanced by Lewis, *do not completely rule out the existence* of smaller circus pathways within the area isolated by the ligatures or crush.

A series of experiments has been performed in our laboratory which confirms and supplements previous observations on blocking the circus pathway.

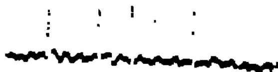




Figure 266 Electrocardiogram of auricular fibrillation in the dog showing no change after freezing of the auricle

Observation 4: Blocking the Hypothetical Circus Path by Burning

In each of four dogs, auricular fibrillation was produced by applying aconitine to the body of the right auricle. Electrocardiograms were taken continuously during the course of the experiment. The right auricle was photographed at 2,000 frames per second before and after two deep burns were inflicted with a hot glass rod. The first burn was made across the intercaval region, extending well up on the wall of the superior and inferior venae cavae. A second burn was executed directly across the body of the right auricle from the auriculo-ventricular groove through the taenia terminalis to the midpoint of the intercaval burn. At autopsy these burns were found to extend to the endocardium.

As shown in Figure 265A, all possible circus paths around either vena cava were effectively blocked by the strategically placed burns. Nevertheless, the simultaneously recorded cinematographs and electrocardiograms clearly show that auricular fibrillation persisted after the burns were inflicted (Figure 265B). The entire right auricle was well visualized on the high speed films. Both large ("L") and minute ("M") contractions were seen to occur on both sides of the burn. The larger contraction waves approached the burned area and terminated; *they did not turn at this point.* No circus movements were visible in any part of the auricle.

Observation 5: Cutting the Hypothetical Circus Path

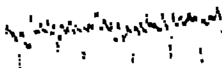
In each of two dogs, the heart was exposed and fibrillation produced by aconitine application. While continuous electrocardiograms were recorded, the right auricle was rapidly cut from the auriculo-ventricular groove to the intercaval region and from the superior to the inferior vena cava. The site of the incision was identical with that of the burn described above. Again auricular fibrillation persisted after complete severance of all possible circumcaval circus paths.

Experiments of this type are necessarily short, since bleeding from the incised auricle cannot be adequately controlled. Nevertheless, auricular fibrillation persisted long enough to allow adequate recordings, thus demonstrating that the electrical phenomena in the fibrillating auricle were independent of the interrupted paths.

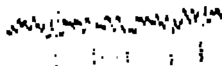
Observation 6: Freezing the Auricle

In each of five dogs, the heart was exposed and fibrillation produced by application of aco-

A. FIRST FOCUS ON APPENDIX OF RIGHT AURICLE



B. SECOND FOCUS PRODUCED ON BODY OF RIGHT AURICLE



C. FIRST FOCUS (APPENDIX) AMPUTATED

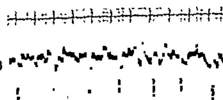


Figure 268 (A) Auricular fibrillation from focus on the appendix of the right auricle (B) A second aconitine focus was produced on the body of the right auricle

Although Scherf was apparently convinced that only one focus was active in aconitine induced auricular fibrillation, he concluded from experiments with acetylcholine that "in some forms of auricular fibrillation, more than one center of rapid stimulus formation is active." [550] Our observations indicate that although it is possible for two or more *foci* to exist simultaneously in the auricles, at a given instant, only one dominates and all others remain latent. If the dominant focus is selectively eliminated, auricular fibrillation may be perpetuated by another formerly dormant focus

From experimental Observations 8 and 9 it is concluded that auricular fibrillation originates from and is perpetuated by a *single* rapidly discharging ectopic focus

SITE OF THE ECTOPIC FOCUS OF CLINICAL AURICULAR FIBRILLATION

Occasionally, electrocardiograms from patients exhibit spontaneous transitions from slower-rate arrhythmias to auricular fibrillation (Chapter XVIII). Conversely, following administration of quinidine to patients with auricular fibrillation, a gradual transition through the slower-rate arrhythmias to normal sinus rhythm is often observed In such instances the (approximate) site of the ectopic focus of the fibrillation can be determined from the configuration of the P′ wave of the slower arrhythmias. As noted in Chapter VIII, an ectopic focus high in either auricle produces an upright P′ wave in leads 1, 2, 3 and AVF, while an ectopic focus low in either auricle produces inverted P′ waves in most of these leads. Through the generosity of our colleagues, we have had the opportunity to study valuable electrocardiograms from 20 patients exhibiting the quinidine-induced transition from auricular fibrillation to auricular flutter. In 14 of these instances the P′ waves of the flutter were inverted in leads 1, 2, 3 and AVF, indicating that the focus of both arrhythmias was low in the

regular waves in the electrocardiogram during fibrillation are a counterpart of large contraction waves which spread from multiple sites for variable distances and in unpredictable directions across the surface of the fibrillating auricles. Such large contraction waves are constantly present in fibrillation and bear no apparent relationship to the location of the experimentally produced ectopic electrical or aconitine focus which is the sole site of formation. Therefore, it would be erroneous to conclude that the variable contour of the oscillations in esophageal electrocardiograms from the fibrillating auricles indicate the presence of many ectopic foci.

The following experiments were performed to determine whether or not more than one independent ectopic focus can be active in the fibrillating auricle.

Observation 8: Fibrillation from Two Successively Produced Foci

In each of three dogs, auricular fibrillation was produced by application of aconitine to the tip of the right auricle appendix. The presence of fibrillation was determined by continuous electrocardiograms from an electrode attached to the body of the right auricle. The appendix containing the aconitine focus was amputated, sinus rhythm was restored almost immediately. A new aconitine focus was produced at the caudal tip of the body of the right auricle and again auricular fibrillation supervened.

These results were duplicated in an experiment in which the first ectopic focus was frozen After normal sinus rhythm returned, and with the first focus still frozen, fibrillation was produced from an ectopic focus at a different site.

The electrocardiographic appearance of the aconitine-produced fibrillation was the same whether the focus was on the tip of the appendix, or at the caudal portion of the body of the right auricle. Similarly it has been observed repeatedly (Chapter XII) that the cinematographic appearance of the auricles during fibrillation remains the same regardless of the site of the focus of origin. Thus, both cinematographically and electrocardiographically, fibrillation appears identical regardless of the location of the experimentally produced ectopic focus. This is in striking contrast to the slower rate arrythmias, in which each contraction and excitation wave starts at the ectopic focus and travels outward in all available directions.

The observation that the electrocardiographic complexes and chaotic contractions of fibrillation disappear when the initiating focus is inactivated and reappear when another focus is produced establishes that these "waves" are not the product of impulses arising from multiple foci which are independently discharging impulses.

Observation 9: Fibrillation from Two Simultaneously Produced Foci

Auricular fibrillation was produced by placing aconitine on the tip of the right auricular appendix; the auricle was photographed. Direct auricular lead electrocardiograms were recorded throughout the experiment. While the bout of fibrillation from the tip of the right appendix was in progress, a second aconitine focus was produced at the caudal border of the body of the right auricle; again cinematographs were taken. The original aconitine focus was then removed by amputating the tip of the right auricular appendix. The electrocardiograms recorded when either focus or both foci were present are identical (Figure 268). The cinematographs also are identical whether recorded when one focus or both foci were present.

Thus the addition of a second ectopic focus during a bout of fibrillation did not affect the cinematographic or electrocardiographic appearance of the arrhythmia. This observation is presented as strong evidence for the view that the fibrillating auricles are not responsive to additional stimuli. Further support for this view is provided by the fact that extrasystoles are never seen in the clinical electrocardiogram during fibrillation and cannot be experimentally produced in the fibrillating auricle by either mechanical or electrical stimulation.

thought to exist (Chapter XIII). The multitude of chaotic oscillations in the fibrillating auricle must maintain a continuous bombardment of the auriculo-ventricular node and under these conditions one would expect an advanced degree of decrement to occur in the auriculo-ventricular pathway. It would therefore appear that the degree of auriculo-ventricular block is much greater than was hitherto believed.

SUMMARY AND CONCLUSIONS

The events leading to the production of auricular fibrillation have been considered. Auricular fibrillation exists when the auricular rate reaches a level at which the auricles are incapable of conducting and contracting in an orderly manner as a unit. The auricular rate at which this event takes place is termed the fibrillation threshold. Cinematographic and electrocardiographic studies of the experimentally produced transition from auricular flutter to fibrillation show that failure of conduction invariably occurs before the increasing auricular rate reaches the fibrillation threshold. This phenomenon is due to incomplete conduction recovery during the shortened diastolic rest period at extremely rapid auricular rates. The hypothesis that auricular fibrillation represents an advanced degree of conduction failure is supported by the observation that the fibrillation threshold is lowered by agents known to decrease the conductivity of the auricles and raised by agents known to increase the conductivity of the auricles.

Factors which predispose to the production of auricular fibrillation include increased vagal tone, auricular distention, anoxia, heart disease and possibly advanced age. In a significant percentage of instances of clinical auricular fibrillation, none of these factors is discernible.

The mechanism by which auricular fibrillation is perpetuated has been investigated. The invalidity of the circus movement theory is demonstrated by cinematographic and oscillographic studies when combined with information obtained from attempts to block the hypothetical circus path by burning, cutting, freezing or removing one auricle during experimentally produced bouts of fibrillation. Cinematographically and electrocardiographically, the appearance of auricular fibrillation is independent of the site of the ectopic focus and is identical whether one or two experimentally produced foci are present on the auricle. The conclusion is drawn that auricular fibrillation originates from and is perpetuated by a single ectopic focus.

The location of the ectopic focus of auricular fibrillation in a small series of patients has been determined. In the majority of instances the focus was low in the auricles.

The irregular ventricular response during auricular fibrillation may be accounted for by the occurrence of decremental conduction in the auriculo-ventricular conducting system. Oscillographic studies indicate that the degree of auriculo-ventricular block in auricular fibrillation is many times greater than was formerly assumed.

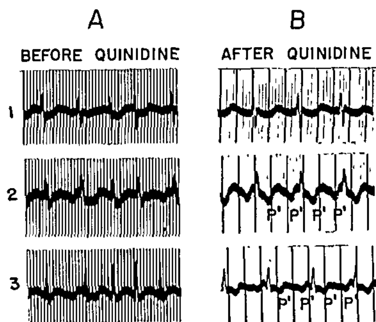


Figure 269 (A) Auricular fibrillation in patient before quinidine.

(B) Conversion of auricular fibrillation to flutter after the patient was given quinidine. The P′ waves are inverted in leads 2 and 3, in this instance, therefore, the ectopic focus is low in the auricle

auricles. In the remaining 6 patients the focus was found to be high in the auricles, (Figures 269 and 270).

The majority of instances of clinical flutter have been found to arise from a focus low in the auricles (Chapter IX). This fact, coupled with the above observation on the site of origin of clinical fibrillation, suggests that foci low in the auricles have a tendency to discharge at a *more rapid rate than those in the cephalic region*. Whether flutter or fibrillation develops would depend upon the rate at which the ectopic focus is discharging and the conductivity of the auricular myocardium.

VENTRICULAR RESPONSE IN AURICULAR FIBRILLATION

Among the more widely accepted explanations of the irregular ventricular response in auricular fibrillation is that originally proposed by Lewis, namely, that the auriculo-ventricular node is stimulated by the highly irregular daughter waves initiating from a main circus path. These impulses were believed to occur at rates near 600 per minute. Since the present study establishes that neither a circus movement nor daughter waves exist, this explanation is no longer tenable.

Lewis and Masters,[377] in a later study of auriculo-ventricular conduction, found that irregular and unpredictable ventricular responses often occur during rhythmic stimulation of the auricle at rapid pre-fibrillatory rates. This phenomenon was attributed to the occurrence of decremental conduction within the auriculo-ventricular conducting system. Direct auricular lead oscillograms indicate that the rate and complexity of the electrical activity of auricular fibrillation is much greater than was previously

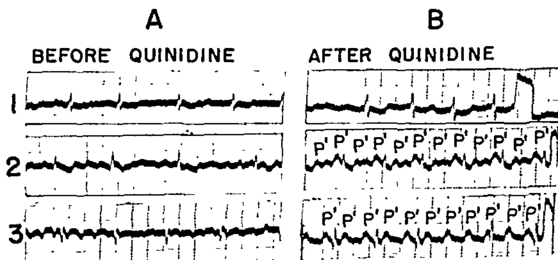


Figure 270. (A) Auricular fibrillation in patient before quinidine

(B) Conversion of auricular fibrillation to flutter following administration of quinidine. The P′ waves are upright in leads 2 and 3, therefore, the focus is high in the auricle.

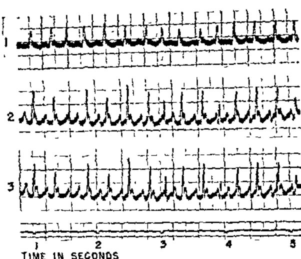


Figure 271 Example of flutter with ventricular aberration produced experimentally in the dog by means of local application of aconitine. The QRS is markedly irregular in both time and shape All auricular complexes are identical except when distorted by ventricular complexes.

FLUTTER WITH CONSTANT REGULAR AURICULO-VENTRICULAR BLOCK

The vast majority of clinical and experimentally produced instances of flutter exhibit constant regular auriculo-ventricular block. Figures 272A and B exemplify this classic or "pure" type of flutter in man. The QRS complexes bear a constant rhythmic relationship to the auricular deflections; usually there is 2:1 auriculo-ventricular block although occasionally the block is of higher degree (4:1 or greater). Two characteristics of the ventricular response are consistently present in this classic type: (1) all QRS complexes originate at corresponding points on the auricular complexes; and (2) all QRS *complexes in a given lead are identical.* The latter circumstance probably depends upon the former; as noted later, in the variety of flutter with irregular auriculo-ventricular block the variation in the shape of the QRS complexes is uniformly associated with variations in their site of origin on the auricular complexes.

FLUTTER WITH VARYING REGULAR AURICULO-VENTRICULAR BLOCK

This relatively rare type of auricular flutter is essentially similar to the classic type described above. Here also, the QRS complexes are all similar in amplitude and configuration and they all originate at corresponding points on the auricular deflection. The only difference

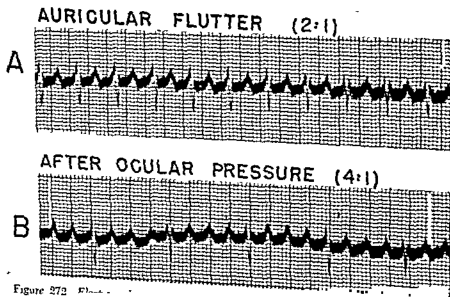


Figure 272 Fl... tone on flutte...
(A) Flut... by the ventri... other flutter wave is masked
(B) Flutter exhibiting 4 1 block resulting after vagal tone was increased by ocular pressure.

CHAPTER XV

Ventricular Aberration in the Auricular Arrhythmias

With Preliminary Observations on the Wolff-Parkinson-White Syndrome

VENTRICULAR aberration occurs in all the auricular arrhythmias but is more common in the auricular premature systoles, flutter and fibrillation.

VENTRICULAR RESPONSE IN AURICULAR FLUTTER

For many years cardiologists have been troubled by an electrocardiographic pattern, frequently encountered clinically, consisting of auricular complexes similar to those of the classic flutter electrocardiogram, but in which the QRS complexes are irregular in time and aberrant in shape. Because of the gross irregularity of the ventricular response, a kinship to fibrillation was assumed or suspected and such "descriptive" terms as "flutter-fibrillation" and "coarse fibrillation" are often applied to the arrhythmia. Other electrocardiographers, recognizing the presence of a basic "flutter" mechanism, designated the pattern "impure flutter." The combination of typical flutter undulations with aberrant ventricular complexes was observed by Lewis in animals and in man, because the tracings exhibited features of both flutter and fibrillation, he interpreted them as representing an intermediate stage between these two arrhythmias. This type of electrocardiographic pattern influenced Lewis' hypothesis of the mechanism of auricular flutter and auricular fibrillation.

The confusion which prevails among electrocardiographers concerning the recognition and designation of this peculiar pattern is witnessed in hospital records and in leading textbooks.[205, 218a, 541] Furthermore, frequently we have seen an interpreter describe some leads of an electrocardiogram as "impure flutter" and other leads of the same tracing as "fibrillation." Such errors in interpretation often are due to differences in voltage of the auricular deflections in various leads. We have observed many tracings in which the amplitude of the auricular deflection was so low that the characteristics of flutter were indistinct and the tracing was read as fibrillation. When such tracings are subjected to close examination or when the voltage of the electrocardiogram is increased, the QRS complexes appear completely irregular in shape and time while the P′ waves are seen to occur at a regular rate and in each lead are uniform in shape.

In the present study, auricular flutter was produced in 40 dogs by means of aconitine application or electric stimulation (Appendix). The electrocardiograms recorded during these experiments included numerous examples of ventricular aberration identical with that described above. One example of ventricular aberration showing irregular R-R intervals is illustrated in Figure 271.

On the basis of experimental observations together with extensive clinical data, we have distinguished three types of ventricular response in auricular flutter. (1) constant regular auriculo-ventricular block; (2) varying regular auriculo-ventricular block, and (3) irregular auriculo-ventricular block and aberrant ventricular complexes. A description of each of these patterns follows.

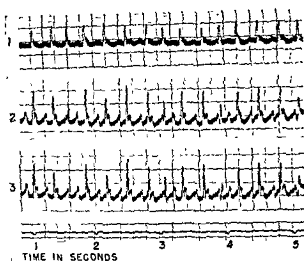


Figure 271. Example of flutter with ventricular aberration produced experimentally in the dog by means of local application of aconitine. The QRS is markedly irregular in both time and shape. All auricular complexes are identical except when distorted by ventricular complexes.

FLUTTER WITH CONSTANT REGULAR AURICULO-VENTRICULAR BLOCK

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FLUTTER WITH VARYING REGULAR AURICULO-VENTRICULAR BLOCK

This relatively rare type of auricular flutter is essentially similar to the classic type described above. Here also, the QRS complexes are all similar in amplitude and configuration and they all originate at corresponding points on the auricular deflection. The only difference

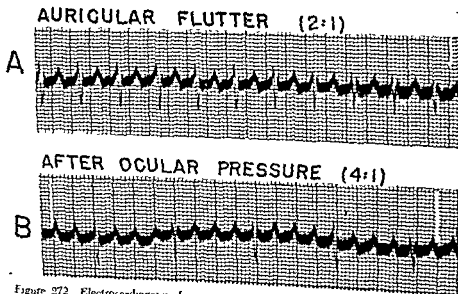


Figure 272. Electrocardiograms from a patient illustrating the effect of increased vagal tone on flutter with constant regular A-V block.

(*A*) Flutter of the "pure" type with 2:1 block. Every other flutter wave is masked by the ventricular complexes.

(*B*) Flutter exhibiting 4:1 block resulting after vagal tone was increased by ocular pressure.

AURICULAR FLUTTER WITH VARYING REGULAR A-V BLOCK

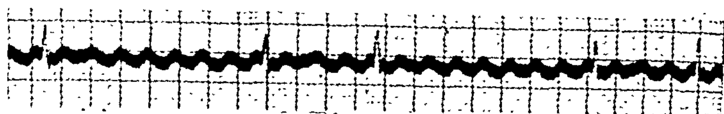


Figure 273. Electrocardiogram from a patient showing varying regular block. In this instance the degree of block alternates between 8:1 and 4:1

consists of a constant variation in the degree of block occurring in the same record (Figure 273). In all instances a simple mathematical relationship exists between the auricular deflections and ventricular responses. This type may bear a superficial resemblance to the "impure" variety but can be distinguished readily by the uniform shape and origin of the ventricular complexes. The varying degree of block probably is due to varying vagal tone.

FLUTTER WITH IRREGULAR AURICULO-VENTRICULAR BLOCK AND ABERRANT VENTRICULAR COMPLEXES

The third type of ventricular complex observed in our study is irregular in rate and variable in configuration. As previously noted, this pattern heretofore has been termed "impure flutter," or "flutter-fibrillation." These names are misleading, as the auricular deflections associated with the irregular ventricular response are identical with those in the classic flutter electrocardiogram. To avoid further confusion in terminology, therefore, it is suggested that the term "auricular flutter with ventricular aberration" be adopted as a suitable designation for this pattern. Terms such as "impure flutter" and "flutter-fibrillation" could then be eliminated from the nomenclature.

Careful study of electrocardiograms of spontaneous and experimental auricular flutter with ventricular aberration reveal the constant presence of several characteristics:

(1) Those QRS complexes which arise from corresponding points on the auricular complexes are uniform in shape and amplitude. All QRS complexes arising from the same part of the P′ waves are identical and all those arising from corresponding points on the Ta waves are identical (Figure 274).

(2) The ventricular responses occur at an irregular rate, that is, the time intervals between the inscription of ventricular complexes bear no simple mathematical relation to one another. Hence the auriculo-ventricular block cannot be described as 2:1, 3:1 etc. (Figure 275). One unusual variation of this type is seen in Figure 276 in which the changes in the P′-R and the *Q-Q intervals are cyclic.*

(3) The QRS complexes vary in shape. Sometimes the QRS complexes are so broad that they suggest a diagnosis of ventricular premature systoles, bundle branch block or idioventricular rhythm. In no instance has a uniform relationship between the degree of QRS aberration and the length of the preceding period of diastole been noted. The variation in configuration of the QRS complexes may be more apparent in some leads than in others (Figure 275).

(4) The QRS complexes originate at different locations on the P′ and Ta waves; that is, the interval between the beginning of the auricular cycle and the onset of the ventricular response is variable (Figure 275). The variation of the P′-R intervals is sometimes of the Wenckebach type, as noted by Langendorff and his co-workers.[40]

(5) The auricular complexes occur at a regular rate and in a given lead they are uniform in configuration and amplitude (Figures 274 and 275). This regularity in shape, size and

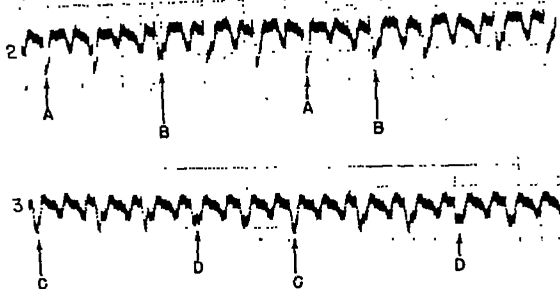
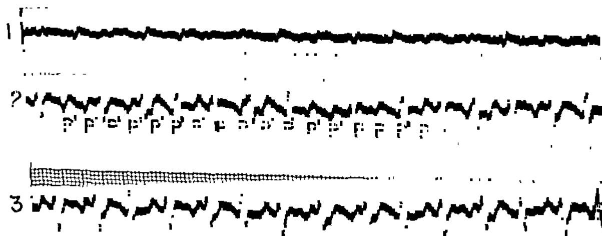


Figure 274 Flutter with ventricular aberration in a patient Carefully compare the configuration of the ventricular complexes In lead 2 the configuration of complex B is different from that of complex A, note that when ventricular complexes *arise from* corresponding points on the auricular complexes they have the same configuration. The same conclusions can be drawn regarding complexes C and D in lead 3



Fig[illegible] abe[illegible] patient The pattern in lead 1 resembles fibrillation In lead 2 the flutter deflections can be seen with ease From lead 3 one might diagnose "coarse" fibrillation with marked ventricular aberration. No simple mathematical relationship between the auricular and ventricular complexes is apparent.

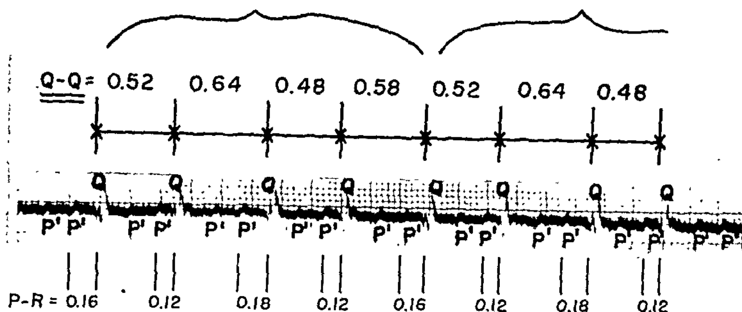


Figure 276. A peculiar case of recurring alternation of the QRS complexes in cycles of 4 beats. Such repetitive responses are rare.

timing of the P′ waves serves to differentiate auricular flutter with ventricular aberration from auricular fibrillation.

It should be emphasized that the variations of the QRS complexes cannot be entirely explained by an electrical summation of the auricular and ventricular complexes.

The type of auricular flutter with ventricular aberration may be present throughout a tracing or it may alternate regularly or irregularly with the classic type of flutter. These alternations occur in both human and experimental subjects.

The observations described above are inconsistent with the view that this interesting electrocardiographic pattern contains elements of both flutter and fibrillation. The regular rate and configuration of the auricular deflections is diagnostic of tachycardia and inconsistent with fibrillation.

CLINICAL CONSIDERATIONS

Classic instances of auricular flutter often are diagnosed or suspected without the aid of an electrocardiogram. Clinical recognition of flutter with aberration is not feasible because the grossly irregular ventricular response, the character of the heart sound, and the character of the peripheral pulse so closely resemble those of auricular fibrillation. The electrocardiogram should render the diagnosis possible in most instances. If the auricular deflections are too small to permit positive identification of the arrhythmia, special auricular leads may be employed to increase the amplitude of the auricular deflections. In many instances it is also necessary to double the voltage on the electrocardiogram. The demonstration of regularly spaced undulations of uniform configuration will establish the diagnosis (Figure 277).

Flutter with ventricular aberration must be differentiated from instances in which the cardiac rhythm alternates rapidly between auricular flutter and fibrillation (Chapter XIII). Clear records with large auricular deflections facilitate the differentiation between these two types of arrhythmia. In those instances in which the cardiac rate is at the fibrillation threshold, the regular undulations of flutter alternate with the jagged deflections of fibrillation* (Figure 278). In contradistinction, the auricular deflections of all types of auricular flutter are uniform and regular throughout the tracing (Figures 271, 272, 273, and 274).

* In the oscillogram tracings we have seen a few auricular beats just before the threshold that are somewhat widened and distorted (Chapter XIV, Figure 248).

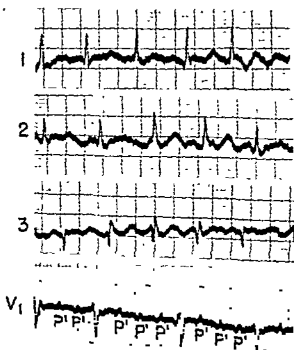


Figure 277 Leads 1, 2 and 3 in a patient simulate fibrillation Lead V_1 shows cleancut P' waves at a rate of 300 per minute The low amplitude of the ...
th

Failure to differentiate flutter with ventricular aberration from flutter with ventricular premature systoles may prevent proper therapeutic management. If the ventricular aberration simulating ventricular *premature systole* appears in the electrocardiogram of a patient under treatment with digitalis, the clinician may consider the patient overdigitalized and discontinue the drug Further administration of digitalis would prove beneficial if the electrocardiographic change represented a transition from classic auricular flutter to the kind with irregular auriculo-ventricular block but not if it represented the development of premature ventricular systoles.

Auricular flutter with ventricular aberration may resemble complete heart block with idioventricular rhythm. An example of complete heart block with idioventricular rhythm is shown in Figure 279.

Electrical alternans is easily misinterpreted as flutter with ventricular aberration. The example of ventricular aberration shown in Figure 280 is due to electrical alternans.

From a therapeutic standpoint, differentiation between the classic type of auricular flutter and the flutter exhibiting ventricular aberration is not important. The basic arrhythmia is the same in both instances and the treatment is identical.

CURREN...
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Since ventricular aberration in the auricular arrhythmias probably involves some disturbance of cardiac conduction, a brief *review of* the generally accepted concept of the *normal* conducting system is in order.

The auricular impulse originating at the sino-auricular node is transmitted through auricular muscle to the auriculo-ventricular *node. At the* auriculo-ventricular node a considerably delay is presumed to occur, following which the impulse is rapidly conducted down the bundle of His and its branches on the endocardial surface of the ventricles to the terminal endings of the conduction system (Purkinje fibers), which are in intimate contact with the myocardium. The ventricles receive the impulse and contract without delay.[41, 848] The position and nature of

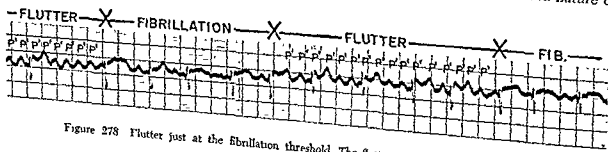


Figure 278 Flutter just at the fibrillation threshold The flutter rate is 360 beats per minute.

Figure 279 (A) Auricular flutter with complete heart block (B) Conversion to fibrillation.

Figure 280 Electrical alternans in a patient with flutter. The P'-R interval is constant Such electrocardiograms must be differentiated from flutter with ventricular aberration.

the auriculo-ventricular node and conducting bundles are such that the electrical activity recorded from this area is due to septal depolarization and does not represent the transmission of the impulse down the bundle of His and its branches. No direct method has been devised to determine the times at which the impulse departs from the auriculo-ventricular junction.

The prevailing concept that a delay occurs in the auriculo-ventricular node is based upon the studies of Lewis,[385] who recorded a lapse of 0.05 second between the time of arrival of the excitation wave at the coronary sinus until it was first recorded by his method in the ventricle. From this data the present theory of normal delay at the auriculo-ventricular node

has evolved without further proof. It is obvious that Lewis' experiment showed only that the delay takes place between the auricles and ventricles; direct proof that it occurs at the auriculo-ventricular node is lacking.

Several theories have been advanced to explain the occurrence of ventricular aberration in the auricular arrhythmias. The explanation most frequently offered is that the ventricular aberration is due to fatigue of the conducting system. Considerable support for this theory has derived from the observation that the shorter the R-P interval in auricular premature systole, the greater is the ventricular aberration.[38, 479, 635] On the other hand, the invalidity of the theory is suggested by an examination of tachycardia and supertachycardias with rates up to 300 per minute. In these arrhythmias the strain on the auriculo-ventricular node, bundle branches and ventricles is far greater than in any of the auricular arrhythmias such as auricular premature systoles, yet the ventricular complexes may exhibit a normal configuration over a long period of time When aberration of the ventricular complexes does occur, it appears abruptly with the onset of the tachycardia and persists unchanged throughout the paroxysm; at the end of the episode, when fatigue of the conducting system should be at a maximum, the QRS and T complexes return to normal with the first sinus beat. It would thus seem that fatigue in the sense of tiring of the tissue of the conduction system cannot be considered the cause of ventricular aberration.

Another theory attributes ventricular aberration to a superimposition of the auricular and ventricular complexes We have found by actual measurement that ventricular aberration in auricular flutter cannot be explained entirely by an algebraic summation of the auricular and ventricular deflections. Furthermore, aberration of the ventricular T wave could not be due to superimposition

Changes in the ventricular complexes during auricular tachycardia in patients with diseased myocardium are due primarily to myocardial ischemia and are not related to the type of ventricular aberration considered in this study. In patients with coronary insufficiency, depression or elevation of the ST segments and inversion of T waves are often observed with bouts of auricular paroxysmal tachycardia. These changes usually occur gradually in damaged hearts but may begin with the first few beats of a paroxysm. Unlike the ventricular aberration associated with auricular arrhythmias in hearts with adequate coronary circulation, the ventricular changes in myocardial disease persist for some time after the tachycardia has ceased (post-tachycardia syndrome). Similar abnormalities may be seen in electrocardiograms from patients with myocardial disease recorded during exercise. Normal hearts usually show none of these alterations during tachycardia or exercise tests.

A relationship between ventricular aberration and auricular rate is suggested by two constant findings: (1) the degree of aberration varies directly with the degree of prematurity of an auricular premature systole; and (2) when the P'-R intervals are the same, the ventricular complexes are the same. Experimental studies by Decherd and Ruskin on ventricular conductivity recovery curves further support this view.[136]

It can be seen that present theories fail to explain adequately the phenomenon of ventricular aberration In order to elucidate this problem, a study of the Wolff-Parkinson-White syndrome is being undertaken in our laboratory. Although to date only preliminary observations have been made, the evidence already obtained suggests a new hypothesis concerning the mechanism of the syndrome. In the following sections these observations and the hypothesis derived from them are presented.

THE WOLFF-PARKINSON-WHITE SYNDROME

Preliminary Observations

The characteristic electrocardiographic abnormalities of the Wolff-Parkinson-White syndrome are as follows: (1) variable shortening of the P-R interval, generally to 0.12 second or less; (2) widening of the QRS complexes,

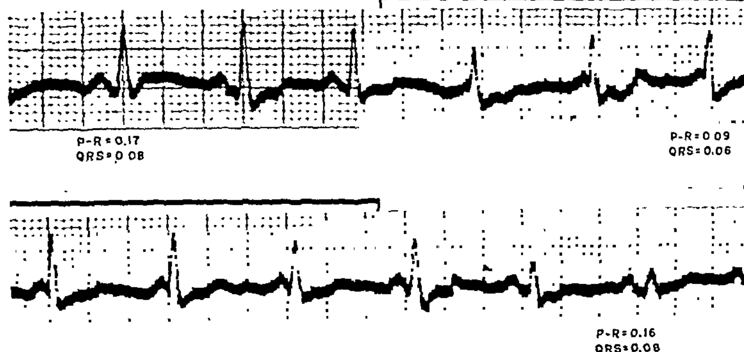


Figure 281. (A) Wolff-Parkinson-White complexes were produced by traction on the pulmonary artery during surgical operation on the lung in a patient with a normal heart. The P-R interval decreased with traction. There is no change in the heart rate.

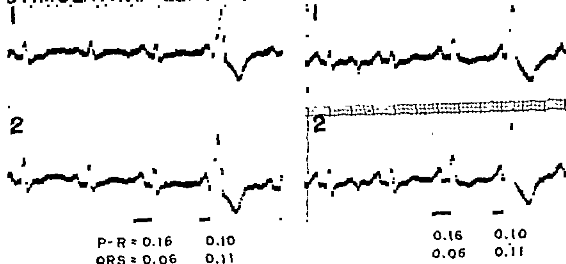


Figure 281 (B) Electrocardiogram recorded during traction on the left auricular appendix in a patient The ventricular complexes resemble those of the Wolff-Parkinson-White syndrome

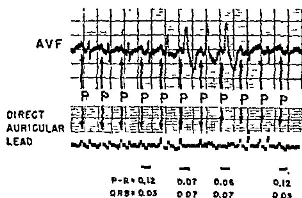


Figure 282 Mechanical stimulation on the body of the right ventricle in a dog Note the run of four ventricular beats resembling Wolff-Parkinson-White complexes

generally to 0.10 second or more; (3) variation in the direction of the QRS complexes so that left or right axis deviation may occur; (4) changes in the ST segment and T waves; and (5) the frequent presence of auricular paroxysmal tachycardia and probably other arrhythmias. The mechanism underlying this syndrome has aroused such extensive speculation that extensive classifications of the many theories have been formulated[331, 473]

During the course of our experimental studies of the auricular arrhythmias, complexes exhibiting all the electrocardiographic features of the Wolff-Parkinson-White syndrome were produced frequently under a wide variety of conditions We have recorded such a pattern from human subjects during surgical procedures in the chest In one patient short runs of Wolff-Parkinson-White aberration were produced whenever the surgeon put traction on the pulmonary artery, upon release of traction the ventricular complexes immediately returned to normal (Figure 281A). In another instance, when the surgeon tugged on the left auricular appendix occasional aberrant beats with short P-R intervals were inscribed (Figure 281B).

Considering the circumstances under which Wolff-Parkinson-White patterns were obtained, their occurrence could not be clearly explained by any previously advanced theory of the mechanism of the syndrome. Therefore, experimental investigation of the problem was instituted.

Observation 1: Production of Wolff-Parkinson-White Complexes by Mechanical Stimulation of the Ventricles

Mechanical Irritation of the Epicardial Surface of the Ventricles: Irritation of the ventricles of the dog with a wooden applicator usually produced ventricular premature beats or "runs" of ventricular tachycardia. By careful manipulation, however, short episodes of Wolff-Parkinson-White aberration were obtained at frequent intervals in almost every animal studied (Figure 282). Long "runs" of Wolff-Parkinson-White aberration were not achieved by this method. The type of aberrant QRS complexes obtained varied with the site of stimulation All five types described by Burch[78] could be obtained. In each of several dogs ventricular complexes of various types were produced by stimulating different parts of the ventricles.

Mechanical Irritation of the Endocardial Surface of the Ventricles: In one experiment a metal rod was pushed through the wall of the right auricle and inserted through the tricuspid opening into the right ventricle. The tip of the rod was pressed firmly against the endocardial surface of the anterior wall of the right ventricle about midway between the apex and the base and about 1½ inches from the septum. Pressure with the rod was maintained for approximately 7½ minutes. Throughout this entire period a "run" of alternating Wolff-Parkinson-White and normal complexes was recorded (Figure 283) Wolff-Parkinson-White complexes have been produced in experimental animals by endocardial stimulation during intracardiac catheterization of dogs.

This type of alternating aberration is not an infrequent clinical finding.[473, 507, 511, 583] We have seen records of two such instances; in one patient the abnormality occurred after exercise (Figure 284), and in the other it developed dur-

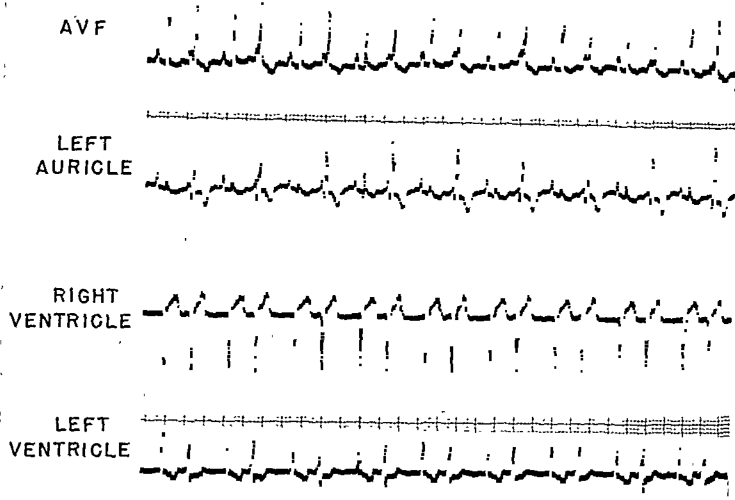


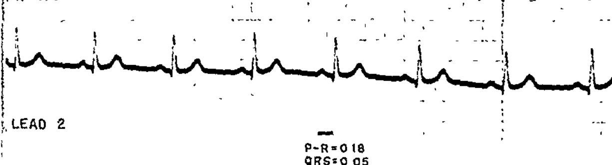
Figure 283. Endocardial stimulation of the anterior wall of the right ventricle in a dog. Every other beat is a Wolff-Parkinson-White complex. The shortening of the P-R intervals is best seen in the direct auricular lead and in lead AVF. The direct lead from the surface of the left ventricle shows interesting variations in the ventricular complexes.

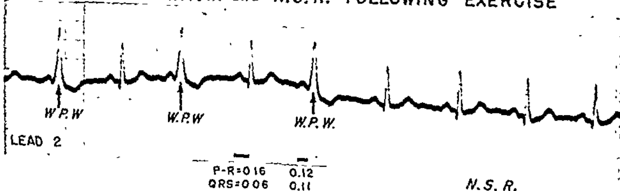
ing tetany (Figure 285). A tracing from the *first patient recorded 16 months after the appearance of the alternating aberration exhibited constant Wolff-Parkinson-White aberration*

Cardiac Catheterization in Man: We have have found a high percentage of Wolff-Parkinson-White complexes in electrocardiograms taken during intracardiac catheterization in man. Similar observations have been made by *Goldman,*[210] *Kossman;*[319] *and Sodi Pallares*[567] The endocardial stimulation by the catheter during this procedure undoubtedly is similar to the endocardial stimulation in experimental animals described above.[567]

Observation 2: Production of Wolff-Parkinson-White Complexes by Chemical Stimulation of the Ventricles

A weak solution of aconitine in benzene (approximately 0.05 per cent) was mixed with Janus Green dye and applied to a small area on the epicardial surface of the ventricles, in no instance was the stained area larger than 1 centimeter in diameter. Aberration exhibiting the electrocardiographic characteristics of the Wolff-Parkinson-White syndrome frequently appeared following application of the chemical (Figure 286). As with mechanical irritation, the direction of the major deflection of the QRS





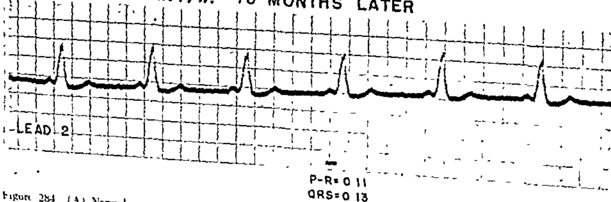


Figure 284 (A) Normal sinus rhythm in a patient
(B) Alternating normal and Wolff-Parkinson-White complexes following exercise in the same patient Note similarity to Figure 283
(C) Sixteen months later the Wolff-Parkinson-White pattern is constant

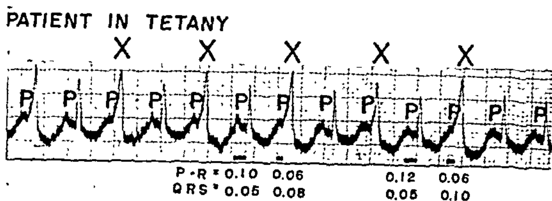


Figure 285. Alternating Wolff-Parkinson-White complexes in a patient with tetany.

complexes varied with the site of stimulation on the ventricles. Stronger solutions of aconitine or repeated application of weak solutions resulted in ventricular premature beats, ventricular tachycardia and ventricular fibrillation.

Occurrence of Auricular Paroxysmal Tachycardia: In at least three of 10 experiments, auricular tachycardia developed following the application of the aconitine and dye solution to the ventricles. In none of these instances was the stain seen on the auricles. In our experience, spontaneous auricular tachycardia does not occur in dogs. Presumably, therefore, the stimulation of the ventricular focus which caused the Wolff-Parkinson-White aberration also produced the auricular tachycardia (Figure 287). In other experiments we have produced auricular flutter and fibrillation under the same circumstances. These arrhythmias are known to occur clinically in conjunction with the Wolff-Parkinson-White syndrome.[474] The mechanism by which auricular paroxysmal tachycardia occurs from a ventricular focus is unknown.

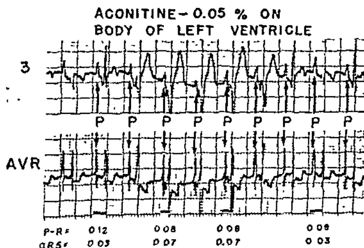


Figure 286. A run of five beats of Wolff-Parkinson-White complexes following local application of a weak solution of aconitine to the body of the left ventricle.



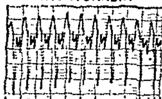


Figure 287. Following the application of aconitine on the ventricles of a dog to produce Wolff-Parkinson-White syndrome, auricular paroxysmal tachycardia also resulted.

It is of interest that during these experiments *all the electrocardiographic* characteristics of the Wolff-Parkinson-White syndrome, including the occurrence of auricular paroxysmal tachycardia, were produced.

Observation 3: Production of Wolff-Parkinson-White Complexes by *Intravenous Administration of* Digitalis Compounds

Electrocardiograms were recorded continuously while digitalis compounds were given intravenously to dogs. In several instances aberration similar to that of the Wolff-Parkinson-

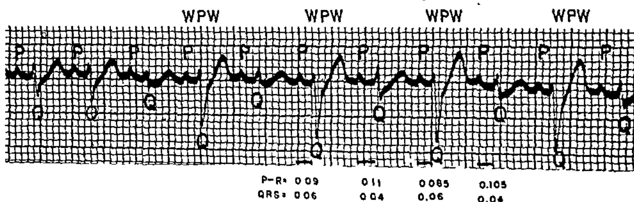


Figure 288 Alternating Wolff-Parkinson-White and normal complexes following administration of a large amount of intravenous ouabain in a dog.

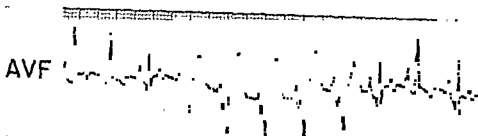


Figure 289 Aconitine on the left ventricular body of a dog after the left auricular appendix had been tied to eliminate the left ventricular inflow tract There is a run of five beats exhibiting Wolff-Parkinson-White aberration

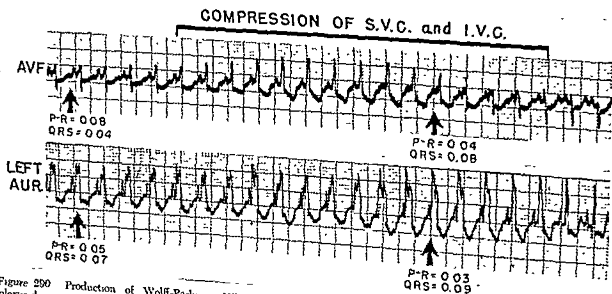
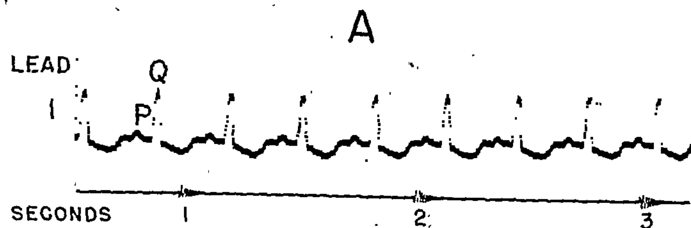
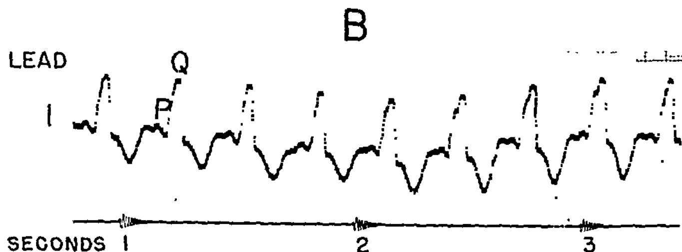


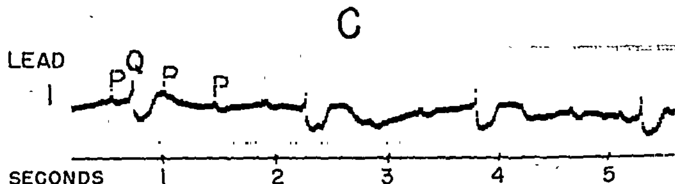
Figure 290 Production of Wolff-Parkinson-White complexes during compression of the superior and inferior venae cavae This experiment rules out the possibility that the ventricular inflow tract acts as a trigger mechanism in the production of the Wolff-Parkinson-White syndrome



a. Lead 1. Normal sinus rhythm. P-R interval, 0 08 second QRS complex, 0 04 second. Recorded at double speed (50 mm /sec)



b. Lead I Same heart. Wolff-Parkinson-White complexes produced by electrical stimulation of the endocardial surface of the right ventricle at the apex Stimulating with direct constant current, 50 volts, 0 5 milliamperes P-R interval, 0.04-0 06 second. QRS complex, 0 07-0 09 second Recorded at double speed (50 mm /sec)



c Lead I Same heart Bundle of His cut Complete heart block present Same stimulating current as in b. No Wolff-Parkinson-White complexes result Recorded at normal speed (25 mm /sec)

Figure 291 The disturbance in ventricular excitation responsible for the Wolff-Parkinson-White complexes in this experiment, therefore, is *mediated through the bundle of His and conduction system*

White syndrome developed following administration of the drug (Figure 288). This finding confirms similar observations made experimentally by Linder in 1940[390]

OBSERVATION 4. THE ROLE OF THE VENTRICULAR INFLOW TRACT

The hypothesis has been advanced that the Wolff-Parkinson-White syndrome is due to an irritable ventricular focus which discharges prematurely when the auricles forcibly eject blood into the ventricles.[286] This hypothesis was tested in the following manner:

Left Inflow Tract: Since the body of the left auricle is noncontractile (Chapter I), normally the left inflow tract is weak. The contractile left auricular appendix was tied off near the base, thus eliminating or markedly reducing the inflow into the left ventricle. Despite this procedure, Wolff-Parkinson-White aberration could be produced by chemical irritation of the left ventricle in the same manner as in Observation 2 (Figure 289).

Right Inflow Tract: By clamping the superior and inferior venae cavae, the inflow tract of the right ventricle was reduced to insignificant proportions In one instance Wolff-Parkinson-White aberration appeared immediately after the venae cavae were clamped (Figure 290).

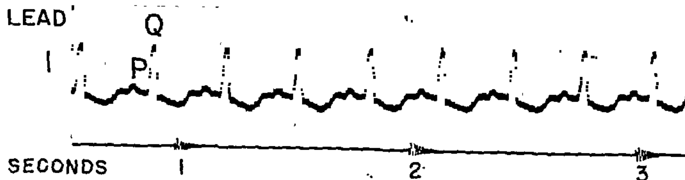
Observation 4 clearly shows that Wolff-Parkinson-White aberration occurs despite complete or partial elimination of the ventricular inflow tract Thus the mechanical force of the inflow tract does not act as a trigger mechanism which causes premature discharge of a hyperirritable ventricular focus in the Wolff-Parkinson-White syndrome.

OBSERVATION 5: RELATIVE INCIDENCE OF WOLFF-PARKINSON-WHITE COMPLEXES AND VENTRICULAR PREMATURE SYSTOLES PRODUCED BY VENTRICULAR STIMULATION

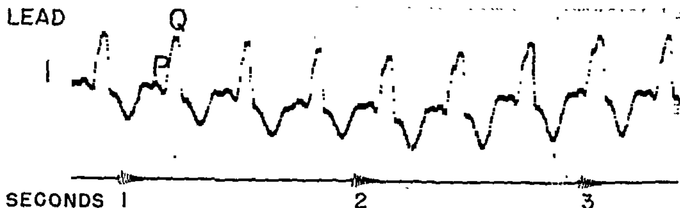
Whether a Wolff-Parkinson-White complex or a premature ventricular systole results from a premature ventricular response depends upon the time during the non-refractory period of the cardiac cycle at which the abnormal response occurs. A premature ventricular response occurring during the period between the crest of the P wave and the onset of the QRS will produce a Wolff-Parkinson-White complex. This period generally occupies about 20 per cent of the non-refractory period at the cardiac rates encountered clinically and in our experimental studies. During the remainder of the non-refractory period – from the end of the refractory period to the inscription of the crest of the P wave or about 80 per cent of the entire non-refractory period – a premature ventricular response produces a ventricular premature systole. According to the law of chance, therefore, random stimulation of the ventricles during the cardiac cycle should produce Wolff-Parkinson-White complexes and ventricular premature systoles in a ratio of approximately 20 to 80 per cent. A statistical analysis of the results of our experiments, in which the ventricles were stimulated at various times throughout the cardiac cycle, has shown that Wolff-Parkinson-White complexes and ventricular premature systoles were produced in a ratio of 48 per cent to 52 per cent.* These statistical findings are signi-

* The abnormal ventricular complexes recorded during the experiments were counted as follows. Each Wolff-Parkinson-White complex preceded and followed by a normal ventricular complex was counted as 1 WPW. Each isolated ventricular premature systole was counted as 1 VPS. "Runs" of ventricular tachycardia starting with a premature systole were counted as 1 VPS "Runs" of Wolff-Parkinson-White complexes were counted as 1 WPW. Over 50 per cent of the "runs" of ventricular tachycardia started with 1 or more Wolff-Parkinson-White complexes, these were counted as 1 WPW. A similar method was used to determine the relative incidence of Wolff-Parkinson-White complexes and ventricular premature systoles produced by intracardiac catheterization in a small series of patients As shown in the Table, the observed ratio in both human and animal studies was significantly different than the ratio predicted on the basis of the law of chance.

Method of Stimulation	*Aconitine and Mechanical (Animals)*		*Catheterization (Man)*	
Average Heart Rate	130		80	
Total No Abnormal Responses	1243		191	
Observed Ratio:				
WPW	601	48%	84	44%
VPS	642	52%	107	56%
Expected Ratio.				
WPW	249	20%	38	20%
VPS	994	80%	153	80%



a. Lead 1. Normal sinus rhythm P-R interval, 0 08 second. QRS complex, 0 04 second. Recorded at double speed (50 mm /sec)



b. Lead 1. *Same heart. Wolff-Parkinson-White complexes produced by electrical stimulation of* the endocardial surface of the right ventricle at the apex. Stimulating with direct constant current, 50 volts, 0 5 milliamperes. P-R interval, 0 04-0.06 second. QRS complex, 0 07-0 09 second Recorded at double speed (50 mm /sec)



c Lead 1 Same heart Bundle of His cut Complete heart block *present Same stimulating current as in b* No. Wolff-Parkinson-White complexes result Recorded at normal speed (25 mm /sec).

Figure 291 *The disturbance in ventricular excitation responsible for the Wolff-Parkinson-White* complexes in this experiment, therefore, is mediated through the bundle of His and conduction system

White syndrome developed following administration of the drug (Figure 288). This finding confirms similar observations made experimentally by Linder in 1940.[398]

Observation 4: The Role of the Ventricular Inflow Tract

The hypothesis has been advanced that the Wolff-Parkinson-White syndrome is due to an irritable ventricular focus which discharges prematurely when the auricles forcibly eject blood into the ventricles[286] This hypothesis was tested in the following manner:

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Right Inflow Tract: By clamping the superior and inferior venae cavae, the inflow tract of the right ventricle was reduced to insignificant proportions. In one instance Wolff-Parkinson-White aberration appeared immediately after the venae cavae were clamped (Figure 290).

Observation 4 clearly shows that Wolff-Parkinson-White aberration occurs despite complete or partial elimination of the ventricular inflow tract Thus the mechanical force of the inflow tract does not act as a trigger mechanism which causes premature discharge of a hyperirritable ventricular focus in the Wolff-Parkinson-White syndrome

Observation 5: Relative Incidence of Wolff-Parkinson-White Complexes and Ventricular Premature Systoles Produced by Ventricular Stimulation

Whether a Wolff-Parkinson-White complex or a premature ventricular systole results from a premature ventricular response depends upon the time during the non-refractory period of the cardiac cycle at which the abnormal response occurs. A premature ventricular response occurring during the period between the crest of the P wave and the onset of the QRS will produce a Wolff-Parkinson-White complex. This period generally occupies about 20 per cent of the non-refractory period at the cardiac rates encountered clinically and in our experimental studies. During the remainder of the non-refractory period – from the end of the refractory period to the inscription of the crest of the P wave or about 80 per cent of the entire non-refractory period – a premature ventricular response produces a ventricular premature systole. According to the law of chance, therefore, random stimulation of the ventricles during the cardiac cycle should produce Wolff-Parkinson-White complexes and ventricular premature systoles in a ratio of approximately 20 to 80 per cent. A statistical analysis of the results of our experiments, in which the ventricles were stimulated at various times throughout the cardiac cycle, has shown that Wolff-Parkinson-White complexes and ventricular premature systoles were produced in a ratio of 48 per cent to 52 per cent.* These statistical findings are signi-

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patients. A [illegible]
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the ratio p[illegible] [illegible] of the law of chance.

Method of Stimulation	Aconitine and Mechanical (Animals)		Catheterization (Man)	
Average Heart Rate	130		80	
Total No. Abnormal Responses	1243		191	
Observed Ratio·				
WPW	601	48%	84	44%
VPS	642	52%	107	56%
Expected Ratio				
WPW	249	20%	38	20%
VPS	994	80%	153	80%

ficantly different from those predicted on the basis of the law of chance and, if accurate, indicate that the occurrence of Wolff-Parkinson-White complexes in our experiments represents a fundamental physiologic phenomenon.

Observation 6: Production of Wolff-Parkinson-White Complexes by Stimulation with Direct Electrical Current

The Wolff-Parkinson-White complexes were produced by non-interrupted direct current in 20 dogs. An insulated cathodal electrode was inserted through a purse-string opening made in either the right or left auricle and passed through the respective auriculo-ventricular valve until it was in contact with the endocardial surface of the ventricle. With the stimulating electrode in place, the current was turned on and gradually increased while simultaneous direct auricular, ventricular and selected limb lead electrocardiograms were recorded. Successful results were obtained most frequently when the electrode was applied to the endocardium at or near the apex of the right ventricle.

With voltages ranging from 15 to 75 volts and a current flow between 0.25 and 1.5 milliamperes, the following results were obtained: (1) In each of 8 dogs continuous runs of Wolff-Parkinson-White complexes were produced. These persisted until the strength of the stimulating current was altered (Figures 291A and B). (2) In 10 dogs, runs of single Wolff-Parkinson-White complexes alternating with normal ones were obtained which persisted as long as the stimulation was continued. (3) Cyclic runs of multiple Wolff-Parkinson-White complexes, alternating with runs of normal complexes, were obtained in at least five dogs.

When the stimulating current was increased (80 to 125 volts; 2 to 3 milliamperes), runs of Wolff-Parkinson-White complexes were obtained that passed into ventricular tachycardia. Weak stimulation with direct current occasionally produced Wolff-Parkinson-White complexes with only slight changes in the P-R interval and the QRS complexes in the direct auricular and ventricular leads and no change in the limb leads.*

That the ventricles, in these experiments, were being driven by the auricles was demonstrated in the following manner. When typical complexes were produced by electrical stimulation, the heart rate could be increased or slowed by artificially changing the temperature of the S-A node. The abnormal ventricular response continued to follow the auricular complex, thus ruling out the presence of idioventricular rhythm or ventricular tachycardia.

In our experience, stimulation by non-interrupted direct current is the best method for initiating the Wolff-Parkinson-White syndrome since by this method, (1) there is little distortion of the record by the current, (2) the degree of stimulation is more accurately controlled, and (3) the tracings are similar to those of human subjects.

Observation 7: Role of Normal A-V Conduction in the Pathogenesis of Wolff-Parkinson-White Complexes

Indirect evidence has been given (Observations 1-6) that the Wolff-Parkinson-White complexes do not result from conduction through anomalous A-V connections. The impulse would therefore seem to travel in some abnormal manner over the normal conduction system. In order to obtain direct proof the following experiment was performed.

In each of seven dogs, runs of Wolff-Parkinson-White complexes were produced by direct current stimulation as in Observation 6. A small ball-tipped cautery, inserted through a second purse-string opening in the right auricle and passed through the tricuspid valve, was used to sever the bundle of His Complete heart block resulted. Attempts were then made to produce Wolff-Parkinson-White complexes in exactly the same manner as before. *In no in-*

* It is possible that clinical cases exhibiting minor electrocardiographic abnormalities are not diagnosed as the Wolff-Parkinson-White syndrome since their only recognized manifestation may be paroxysmal tachycardia Cases exhibiting the classical electrocardiographic pattern are probably well marked examples of the syndrome, since distortion must be severe to be reflected in the limb leads.[7]

stance did these complexes occur. Idioventricular beats occurred but did not follow the P wave except by chance (Figure 291C). This experiment demonstrates that the Wolff-Parkinson-White complexes cannot be produced without an intact bundle of His. If anomalous A-V bundles transmitted the impulse from the auricles to the ventricles and were responsible for the aberrant ventricular complexes, severing the bundle of His should not have interfered with the production of Wolff-Parkinson-White complexes.

DISCUSSION

The theory has been advanced that premature excitation of the ventricles in the Wolff-Parkinson-White syndrome is caused by a short-circuit of the auricular impulse along aberrant muscle bundles between the auricles and ventricles[670] This concept has received strong support as a result of the excellently conducted experiments of Butterworth and Poindexter.[81, 85] These workers picked up the auricular impulse with a direct lead, amplified it several thousand times, and after a suitable selected delay fed it into the ventricles, the resulting electrocardiograms exhibited typical Wolff-Parkinson-White complexes. One cannot deny that auriculo-ventricular muscular bridges such as the bundle of Kent have been found in some instances of Wolff-Parkinson-White syndrome as well as in patients without the syndrome[473, 677] The mere presence of these structures does not prove that they conduct aberrant impulses and are responsible for Wolff-Parkinson-White aberration A complete histologic examination of all the auriculo-ventricular junctional tissue of a human heart could easily consume several months To fully evaluate the reported bundles in some patients exhibiting the Wolff-Parkinson-White syndrome, control studies would have to be done to establish that similar bundles are absent in normal hearts An investigation of this type sufficiently extensive to give statistically significant results would appear most formidable

On the other hand, the validity of the theory of conduction through anomalous anatomical pathways can be questioned in other ways. If the Wolff-Parkinson-White complexes were produced by transmission of the auricular impulse over anomalous auriculo-ventricular pathways, the production of these complexes by stimulating widely separated points in either ventricle makes it necessary to assume that the anomalous bundles have connections with the ventricular myocardium as extensive as those of the normal conduction system. Such connections are not known to exist.

Having adduced evidence that conduction over anomalous pathways was not responsible for the Wolff-Parkinson-White complexes, we interpreted our experimental results to mean that conduction probably proceeded in some abnormal manner over the normal pathways. When this hypothesis was tested it was found that Wolff-Parkinson-White complexes could not be produced after the bundle of His was cut. In other words, premature ventricular excitation depended upon the intact *normal* conducting system. Thus, the Wolff-Parkinson-White syndrome may be properly considered a physiologic disturbance rather than an anatomic one, just as aberration during auricular arrythmias is an obvious physiologic disturbance.

In order to determine just what this disturbance is we must first consider certain features of normal auriculo-ventricular conduction. The experimental work on the subject to date does not fully explain where the delay in normal auriculo-ventricular conduction occurs. The difficulty arises because there is no way of detecting the impulse as it travels down the conduction system. The excitation impulse is known to pass from the S-A node to the A-V node at such a rate that auricular transmission accounts for a very small part of the normal P-R interval. When the time for transmission of the impulse over the auricles has been taken into account an unexplained interval exists before the first electrocardiographic sign of ventricular depolarization. Most investigators today believe that this portion of the P-R interval, is the result of slow transmission of the impulse through the

A-V node. The impulse is then believed to be rapidly conducted down the bundle of His, the bundle branches, and the Purkinje system and out to the ventricular musculature, where it causes immediate depolarization.

An alternative theory of A-V conduction, known as the "Latency Theory," postulates that the major part of the P-R interval does not necessarily result from the slow passage of the impulses through the A-V node, but rather from a delay at the terminal endings of the Purkinje system. After this peripheral delay has been overcome ventricular depolarization then occurs.

Let us now consider the nature of the Wolff-Parkinson-White syndrome in respect to the above two theories. If the first of these is correct, the delay takes place at the A-V node. Thus in our experiments a disturbance in a ventricular focus must have caused the impulse to be conducted more rapidly through the normal node to the altered point. The disturbed area thus depolarizes early causing the P-R interval to be shortened and the QRS complex to begin prematurely. Depolarization of the rest of the ventricles occurs after the usual A-V node delay. The resulting QRS complex is widened and distorted. This theory, suggested by others in the past, postulates that the bundle of His is a complex structure consisting of many fibers which go to separate parts of the Purkinje system and ventricles. When a point in the ventricle is altered under the conditions of our experiments some type of antidromic disturbance allows certain fibers to conduct the normal impulse at a rate more rapid than normal. The resulting electrocardiographic pattern depends upon which site of the ventricular myocardium is depolarized prematurely. This would explain the wide variety of patterns encountered. Since the A-V node lies partly in the auricle, it is conceivable that the antidromic disturbance is in some manner related to the occurrence of auricular paroxysmal tachycardia

Let us now consider the Wolff-Parkinson-White syndrome if the second theory of A-V conduction is correct. This theory postulates that the normal delay in A-V conduction is at the periphery. The stimulated point in the ventricle shortens the delay allowing premature excitation at that point. Since the area becomes excited immediately upon or shortly after arrival of the impulse the QRS complex begins prematurely and the P-R interval is shortened proportionately. The configuration of the QRS complex and the degree of shortening of the P-R interval are determined by the location of the sensitive site and the degree of prematurity of depolarization. The resulting beat is thus a product of varying proportions of premature and normal ventricular excitation.

The exact mechanism of the Wolff-Parkinson-White syndrome and the ventricular aberration in the auricular arrhythmias cannot be determined at present. Direct evidence concerning what actually takes place is difficult to obtain because the normally conducted A-V impulse cannot be measured. The currents that are obtained over the right septal region are due to septal depolarization and not to the conduction of the impulse through the bundle of His and its branches Until the nature of normal A-V conduction is completely understood we can only speculate on the nature of ventricular aberration associated with the Wolff-Parkinson-White syndrome.*

If our observations can be applied clinically one might prophesy that if a patient exhibiting the Wolff-Parkinson-White syndrome has interruption of the bundle of His, as for instance after myocardial infarction, it would result in the disappearance of this syndrome This, of course, would be final proof that the mechanism of clinical Wolff-Parkinson-White syndrome is identical with that produced in our experiments On the other hand if the aberrant A-V bundle theory is correct, interruption of the bundle of His by disease should not produce complete heart block and should, in fact, make the Wolff-Parkinson-White complexes more bizarre, since the entire activation

* Subsequent experiments in this laboratory have demonstrated that the normal delay takes place at the A-V node. Evidence has been presented that in the Wolff-Parkinson-White syndrome and ventricular aberration, the normal delay has been overcome. The essential disturbance would appear to be accelerated conduction through the A-V node

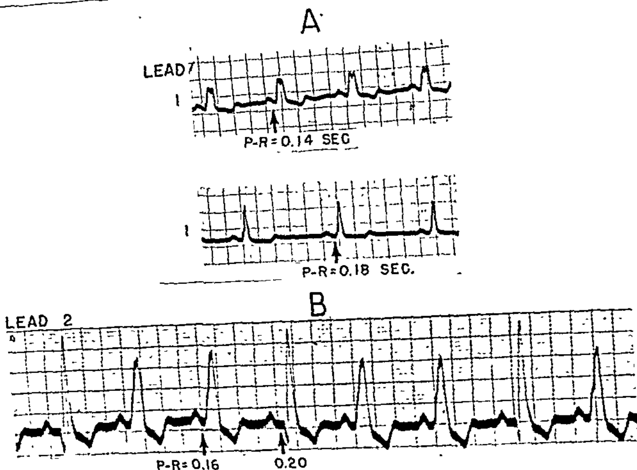


Figure 292 (A) Functional left bundle-branch block in ... rhythm in lower tracing. ... sinus rhythm

(B) During the very wide QRS complexes preceding, the P-R intervals are 0 16 second. The narrower and sharper ... by P-R intervals which have ... patients exhibited ... (From William Dressler Am H J, 29 ..., courtesy of C V. Mosby Co, St. Louis)

of the ventricles should take place through the aberrant A-V pathway.

The ventricular aberration characteristic of the syndrome exhibits many similarities to that seen in the auricular arrhythmias (1) the QRS complex is distorted or widened, (2) the shape of the QRS complex is directly related to the length of the P-R or P′-R interval, (3) many patterns of aberration may occur, (4) the T waves are altered, and (5) when complete heart block is present no ventricular aberration exists The last observation is supported by 6 cases of flutter with complete heart block in which no ventricular aberration was noted (Figures 128, 133, and 142)

We have observed that shortening of the P-R interval may occasionally occur in bundle-branch block. We have seen electrocardiograms showing normal and bundle-branch block complexes alternating in rapid sequence in which the P-R intervals occasionally were shortened during the block. Similar shortening of the P-R interval has been observed in partial bundle-branch block (Figure 292).

The similarities between Wolff-Parkinson-White aberration and the ventricular aberration associated with the auricular arrhythmias, with alternating bundle-branch block and with certain intraventricular blocks suggest the existence of a broad general principle of ventricular aberration applicable to all these and possibly other disorders. It is apparent that much is yet to be learned concerning the conduction system, that the prevailing theories of normal au-

A-V node. The impulse is then believed to be rapidly conducted down the bundle of His, the bundle branches, and the Purkinje system and out to the ventricular musculature, where it causes immediate depolarization.

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ing all the criteria of the Wolff-Parkinson-White syndrome could be reproduced by stimulation of ventricular foci far removed from the auriculo-ventricular node further establishes the fact that the syndrome is not caused by conduction via an aberrant auriculo-ventricular muscle bundle.

Until more information concerning the normal conduction system is known the true nature of ventricular aberration in the auricular arrhythmias, the Wolff-Parkinson-White syndrome and other cardiac disorders will be purely speculative.

riculo-ventricular conduction and of the Wolff-Parkinson-White syndrome are as yet unproven, and that the theories suggested by our preliminary observations must be subjected to more critical examination than has been done in this present study. Ultimately, complete understanding of ventricular aberration will depend upon the clarification of the normal conduction mechanism of the heart.

SUMMARY AND CONCLUSION

The ventricular response associated with experimentally produced auricular flutter in the dog and the spontaneously occurring disease in man has been studied. Three types of ventricular response in auricular flutter are distinguished: (1) constant, regular auriculo-ventricular block (classic type), (2) varying regular auriculo-ventricular block, and (3) irregular auriculo-ventricular block with aberrant ventricular complexes. Since the auricular deflections of these three types are identical, the common practice of referring to the third type as "impure flutter," "flutter-fibrillation" or "coarse fibrillation" is misleading. The term "auricular flutter with ventricular aberration" is suggested as a more suitable designation for this pattern.

The QRS complexes of auricular flutter with ventricular aberration occur at an irregular rate, vary in configuration and originate at various sites on the auricular deflections. QRS complexes which originate at corresponding sites on the auricular deflections are identical Alternations between regular auriculo-ventricular aberration may occur in a single electrocardiogram.

Auricular flutter with ventricular aberration is differentiated from auricular fibrillation by the presence of regularly spaced auricular deflections in the electrocardiogram. Special auricular leads or doubling of the voltage on the electrocardiogram may be necessary to obtain auricular deflections of sufficient amplitude to permit definite differentiation. Other electrocardiographic patterns which resemble auricular flutter with ventricular aberration include auricular flutter with ventricular premature systoles, complete heart block with idioventricular rhythm, and auricular flutter with electrical alternans.

Prevailing theories concerning the mechanism of normal auriculo-ventricular conduction and of ventricular aberration in the auricular arrhythmias are based on indirect and inconclusive evidence. Aberration in auricular flutter takes place through the normal conduction system since it is not present when complete heart block exists.

In a preliminary series of experiments, short episodes of ventricular aberration exhibiting all the electrocardiographic features of the Wolff-Parkinson-White syndrome have been produced in dogs by mechanical and chemical stimulation of multiple sites on the ventricles, by direct, non-interrupted cathodal current applied to the ventricular endocardium, by intravenous administration of digitalis, and by clamping the venae cavae. Occasional Wolff-Parkinson-White complexes also were obtained by epicardial stimulation. The records obtained by stimulation with direct current proved most advantageous for the purposes of study. Wolff-Parkinson-White complexes were produced fortuitously in man during surgical procedures in the chest by tugging on the pulmonary artery or the left auricular appendix. Wolff-Parkinson-White aberration was obtained by mechanical stimulation of the ventricles after drastic reduction or elimination of the left and right ventricular inflow tracts, indicating that the mechanical force of the inflow tract does not precipitate the premature ventricular response in the syndrome by setting off an irritable ventricular focus.

In 7 consecutive experiments the Wolff-Parkinson-White complexes produced by cathodal endocardial stimulation was eliminated when the bundle of His was severed producing complete heart block. This clearly demonstrates that the Wolff-Parkinson-White syndrome in our experiments resulted from abnormal conduction through the normal conducting system and not through anomalous A-V pathways. The observation that an electrocardiographic pattern fulfill-

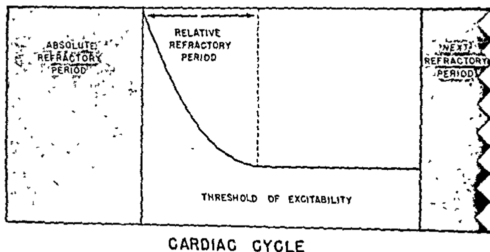


Figure 293. Hypothetical figure summarizing the approximate relationship of the absolute and rel... period ... fractor... current is required to excite.

lation. Love[394] demonstrated conclusively that excitation, if sufficiently premature, takes place with little or no conduction of the excitatory process. The term decremental conduction has been used to describe those instances in which an excitation wave is conducted for a short distance with gradual and premature termination of the wave (a fading-out effect) Love's observation indicates that conduction recovery takes place after the absolute refractory period has ended* (see Figure 256)

The Significance of Auricular Rate

The factor of auricular rate has not been given adequate consideration in many investigations of the effects of anti-arrhythmic drugs When the duration of the relative refractory period is determined at an auricular rate of 100 per minute, the figure obtained is quite different from that obtained at an auricular rate of 500 per minute. Similarly, the electrically determined threshold of auricular excitability at an auricular rate of 100 is less than the threshold of excitability of the same auricle beating at a rate of 500 Finally, the rate of conduction at an auricular rate of 100 is greater than at an auricular rate of 500 The effects of a drug administered when the auricles are beating at a rate of 100 may be not only quantitatively different but also qualitatively different than the ...

... results when determinations of pharmacologic actions made in an auricle beating at a given rate are applied to auricles beating at significantly faster or slower rates.

The Absolute Refractory Period

Early investigators sought unsuccessfully to determine the duration of the absolute refractory period by stimulating with currents several times stronger than threshold Most such studies were carried out by stimulating at one point and recording the electrical events produced at another point in the heart.* Since excitation

* The concept recently advanced by Wedd, Blair and Gosselin suggests that recovery of conductivity and excitability occur simultaneously during the relative refractory period If such a relationship exists, it would account for the generally recognized overlapping between studies of various phases of the recovery process which follows the absolute refractory period

* Various modifications of the following method were generally used The heart rate is maintained constant by rhythmic electrical stimulation. Extra shocks delivered through another electrode are administered at various intervals after the preceding rhythmic stimulus The first extra shock that produces a conducted response is considered to mark the end of the absolute refrac...

CHAPTER XVI

Pharmacologic Considerations in the Auricular Arrhythmias

PREVAILING concepts of the *mechanism of action* of drugs effective in terminating auricular flutter and fibrillation are based primarily on *the circus movement theory. The drugs are* presumed to abolish the "excitable gap" in the circus pathway which, according to the theory, is *present during flutter or fibrillation.* The effect of therapeutic agents in auricular paroxysmal tachycardia is attributed to a similar mechanism by *those* workers who believe that tachycardias also involve a circus movement

Since the observations reported in this monograph indicate that the circus movement theory is untenable, a re-evaluation of the action of anti-arrhythmic agents is required. A thorough investigation of the pharmacologic action of these agents is beyond the scope of the present study. Nevertheless, the pertinent literature has been reviewed and several cinematographic, electrocardiographic and oscillographic observations have been made in an effort to achieve a new concept of the mode of action of the two most commonly used anti-arrhythmic agents, quinidine and digitalis. Pending further elucidation of the complex problems involved, this material is discussed in the present chapter in the hope that it will prove of interest to persons especially concerned with the pharmacologic aspects of therapy. Since much of the pharmacologic discussion is of a technical nature and not essential to an understanding of practical therapy, all material of clinical interest has been included in the summary at the end of the chapter for the benefit of the general reader. Practical aspects of the therapy of the auricular arrhythmias are discussed in Chapter XVII.

COURSE OF EVENTS IN THE CARDIAC CYCLE

The physiologic events of the cardiac cycle *taking place after direct stimulation of the heart* are dependent on the excitability, conductivity and contractility of heart muscle. A state of excitability exists *immediately following* the absolute refractory period and prevails until the end of the cardiac cycle. The threshold of auricular excitability gradually falls from a maximum at the onset of the exictable phase to a level which remains fairly constant for the remainder of the cycle (*Figure 293*). The interval between the end of the absolute refractory period and the leveling off of the threshold of auricular excitability has been arbitrarily defined as the relative refractory period; the strength of the stimulating current required to excite the muscle declines throughout the relative refractory period and reaches a minimum at its termination. When the cardiac cycle is extremely short, as occurs at very rapid cardiac rates, the threshold of excitability fails to reach a constant level and the relative refractory period persists until the onset of the succeeding cycle. During the absolute refractory period no stimulating current, regardless of its strength, evokes a response.

When the heart responds to a stimulus, excitation, conduction and contraction normally follow. Conduction *is said to take place* when the excitation and contraction waves spread across the heart. The fact has long been known that the rate of conduction is slowed when the cardiac cycle is shortened by premature stimulation as in premature systole, or by rapid stimu-

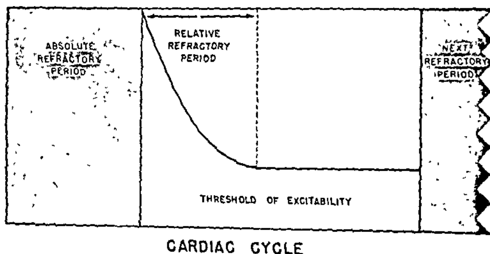


Figure 293 Hypothetical figure summarizing the approximate relationship of the absolute and relative ... period no ... fractory pe... current is required to excite

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The Absolute Refractory Period

Early investigators sought unsuccessfully to determine the duration of the absolute refractory period by stimulating with currents several times stronger than threshold. Most such studies were carried out by stimulating at one point and recording the electrical events produced at another point in the heart.* Since excitation

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* Various modifications of the following method were generally used The heart rate is maintained constant by rhythmic electrical stimulation Extra shocks delivered through another electrode are administered at various intervals after the preceding rhythmic stimulus The first extra shock that produces a conducted response is considered to mark the end of the absolute refractory ... ceding sh...

may take place under the stimulating electrode with little or no conduction of the excitatory process to the distal electrode, this method is unsatisfactory for determining the end of the absolute refractory period. It was found that the results obtained were particularly inaccurate when conduction recovery was delayed by drugs or other factors.

An alternative method which avoids these difficulties was devised by Love [394] who used an ingenious stimulator which permits determination of the end of the absolute refractory period without the error introduced by decremental conduction.* By this method, excitation is demonstrated to have taken place by the failure of a second stimulus to excite.

Lewis and Drury [378] suggested that the figure obtained by the distal electrode method be termed the "effective refractory period." The effective refractory period thus consists of the absolute refractory period plus a portion of the relative refractory period the length of which varies inversely with the rate of conduction recovery in the muscle under study and directly with the distance between the stimulating and recording electrodes. It is therefore obvious that the demonstration of a lengthened or shortened effective refractory period may be dependent on either or both of two factors which bear no relation to the absolute refractory period. Although the term "effective refractory period" has enjoyed wide usage, in our opinion the concept is of doubtful worth and continued use of the term is confusing.

Other investigators have related the length of the absolute refractory period to the Q-T interval. Such methods have proved accurate since errors involving changes in conduction recovery apparently are eliminated Macleod [421] found in the isolated turtle heart that the Q-T interval is directly related to the absolute refractory period, there being only a slight difference in their actual measurements. Wedd and his associates,[43, 620, 621] in a study of isolated strips of turtle auricles and ventricles, concluded that measurements of the Q-T interval and the absolute refractory period are identical. However, many factors influencing the Q-T intervals in the *clinical* electrocardiogram are not related to changes in the absolute refractory period.[506] Lengthening of the Q-T interval in the clinical electrocardiogram does not necessarily indicate that the absolute refractory period has been lengthened, although there is a close relationship in the muscle strip.

Part I

THE PHARMACOLOGY OF QUINIDINE

Quinidine is perhaps the best example of an anti-arrhythmic drug It is effective in all the auricular arrhythmias.

Wenckebach introduced quinine into the therapeutics of auricular fibrillation in 1914.[632] This investigator relates an experience with a Dutch merchant who presented himself with a history of frequent attacks of auricular paroxysmal fibrillation. The patient had discovered that quinine taken during his attacks stopped the paroxysms within 20 minutes of their onset. Wenckebach subsequently used this remedy successfully in other cases of auricular fibrillation. Frey [206] was the first to employ quinidine, the dextro-isomer of quinine, in the therapy of auricular arrhythmias. In an excellent monograph Gold [235] recently summarized clinical ex-

* The heart rate is maintained constant by stimulation at any given rate through one system, while a second system delivers two rapid shocks at progressively decreasing intervals after each rhythmic stimulus All stimulation takes place through one electrode; tracings are recorded from a second electrode near the site of stimulation After five rhythmic shocks (S-1) have been delivered by the first system, the two rapid shocks (S-2 and S-3) separated by a predetermined and fixed interval are administered following the last rhythmic stimulus, thereafter, the interval between the rhythmic stimulus and the two extra shocks is progressively increased The end of the absolute refractory period is marked when S-2 fails to produce an auricular deflection but nevertheless prevents or delays the response to S-3 Thus the duration of the absolute refractory period can be determined by measuring the interval between S-1 and S-2, no measurement of the intrinsic deflection is required

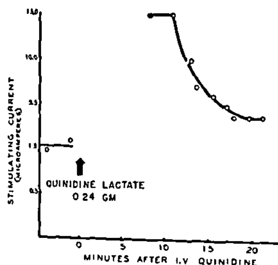


Figure 294. 200 before a tration of qu cles at a rate of 200 is stronger than in the unmedicated animal The effect of quinidine on the threshold of auricular excitability is even greater for more rapid rates Anesthesia, pentobarbital.

perience with quinidine and its present-day usage.

Effect of Quinidine on the Absolute Refractory Period

Investigators prior to 1925 reported a lengthening of the absolute refractory period of cardiac muscle following administration of quinidine More recent work, however, has revealed that when measures are taken to eliminate or decrease error due to decremental conduction or incomplete conduction recovery, quinidine *either shortens or has no effect on the absolute refractory period* [394, 621] This fact has been accepted and confirmed by Lewis [378]

Effect of Quinidine on Auricular Excitability

We have confirmed the observations of other workers [621] that the threshold current required to produce a given auricular rate is raised by quinidine (Figure 294). This increase in the threshold of auricular excitability occurs even at slow auricular rates but is disproportionately greater at rapid rates. Further evidence of the threshold-raising effect of quinidine is provided by the observation that mechanical stimulation such as stroking or pinching the auricles failed to elicit premature systoles after administration of the drug whereas previously extrasystoles were easily produced by this method. Similarly, the application of relatively large amounts of aconitine to sites on the auricles after quinidinization failed to produce arrhythmias. In untreated dogs we have found aconitine unfailingly effective in initiating auricular arrhythmias.

Observation 1: Effect of Quinidine on the Cinematographic Appearance of the Auricles

In each of four dogs, a stimulating electrode was attached to the body of the right auricle adjacent to the entrance of the inferior vena cava. Single shooks from a low frequency pulse generator were sent into the auricle at a rate greater than that from the sinus node, usually from 120 to 300 per minute. Cinematographs were recorded at a film speed of 2,000 frames per second while control electrocardiograms confirmed the presence of auricular tachycardia and flutter. Quinidine lactate, 20 mgm. per kilogram, was given intravenously and motion pictures were taken while the auricle was stimulated *at the same rate* as previously. The films recorded before and after quinidine administration were then projected on the screen simultaneously and adjacent to each other. In both cinematographs the auricular contractions originated at the site of attachment of the electrode and traveled across the auricle. The contraction wave in pictures of the auricle taken after administration of quinidine consistently traveled at a far slower rate than those occurring before the drug was injected. Even after stimulation was stopped and normal sinus rhythm restored, the contraction waves traveled at abnormally slow rates.

Previous investigations on the subject without

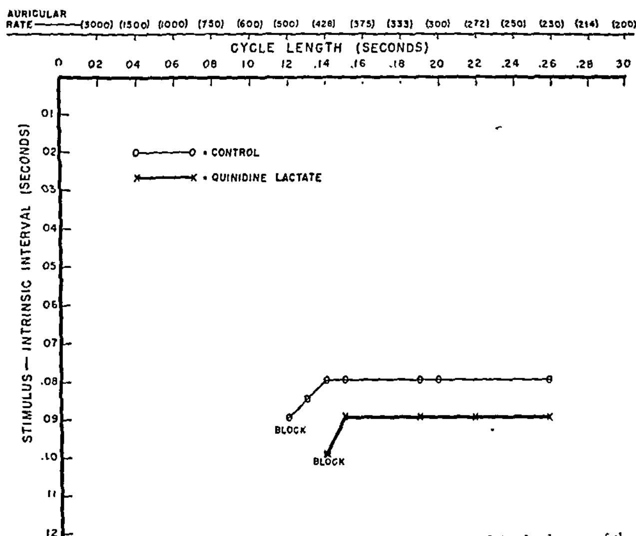


Figure 295 Auricular conduction recovery before and after quinidinization. Ordinates (Y axis) shows stimulus-intrinsic interval (transmission interval) in seconds as measured in direct lead electrocardiograms from left auricle recorded while the right auricle was being electrically stimulated Abscissae (X axis) shows the stimulus-stimulus interval (cycle interval) in seconds All electrode positions were left unchanged for the duration of the experiment For reference the corresponding auricular rate is shown above the stimulus-stimulus interval

Note that following administration of quinidine (10 mgm./Kg.), conduction recovery is delayed and incomplete. (Urethane Anesthesia)

exception have revealed that quinidine slows the speed of the excitation wave.[374, 375, 621] This electrocardiographic observation is confirmed by the visual demonstration that slowing of the rate of conduction of the auricular contraction wave occurs following administration of quinidine. Such slowing of conduction is responsible for the wéll known widening of the P and P′ wave in electrocardiograms from patients treated with quinidine and is a manifestation of the depressing effect of the drug on recovery processes in the auricles.

Observation 2: Effect of Quinidine on Auricular Conduction Recovery

In the present study the effect of quinidine on conduction recovery in the auricles was determined by measuring the rate of conduction at various rates of stimulation. In five dogs with the heart exposed, direct auricular leads were recorded simultaneously from the body of the right auricle and the left auricular appendix. A stimulating electrode from a low frequency pulse generator was attached to the right auricular appendix, and the positions of all electrodes were left unchanged for the duration of the experiment. The auricle was then stimulated by currents several times threshold at rates in gradual increments of 25 to 50 stimuli per minute while continuous electrocardiograms were recorded at double speed (50 millimeters per second). Such records were made in duplicate



Figure 296 Stills from motion picture to demonstrate the effect of quinidine on the auricular myocardium

Upper photograph demonstrates the maximum diastole of auricular fibrillation produced by aconitine in a dog.

Lower photograph shows maximum diastole of auricular fibrillation after quinidine has been administered. Note that after treatment with quinidine the auricle is markedly distended due to loss of myocardial "tone."

before and after the administration of fractional doses of quinidine (5 to 20 mgm. per kilogram body weight). Measurements were made of the stimulus-intrinsic interval * to the nearest 0.01 second in direct leads from the left auricle. Portions of the tracings recorded while the auricular rate was being changed were not used for the calculations.

In the untreated auricle, increasing conduction failure was found to occur as the auricular rate was increased beyond a certain level, as manifested by an increased stimulus-intrinsic interval and a slight splintering and widening of the P' wave (Observation 2: Chapter XIV). After these events became well developed, a further increase in the rate of stimulation produced one of two reactions: (1) variable auricular blocking to the electrical stimulus; or (2) auricular fibrillation. Following the administration of quinidine, these manifestations of reduced conductivity were apparent at all rates of stimulation, becoming more marked at rapid auricular rates. Similarly, the P waves during normal sinus rhythm were wider. Only occasionally was it possible to accelerate the auricular rate sufficiently to produce fibrillation. Figure 295 shows the effect of quinidine on auricular conduction recovery as determined by plotting the stimulus-intrinsic interval against the stimulus-stimulus interval. Since quinidine causes a prolongation of the stimulus-intrinsic interval at all rates studied, conduction recovery following quinidinization is delayed and incomplete (or infinitely delayed). As indicated in Figure 295, after quinidine has been administered it is impossible to drive the auricles as fast as in the unmedicated animal Although the apparent failure of the auricles to respond to more rapid rates of stimulation following quinidine was formerly attributed to a lengthening of the absolute refractory period, Love [394] has presented evidence which indicates that conduction failure may be responsible for this phenomenon.

* This interval includes not only the conduction time but also the latent period which introduces a negligible and constant error.

Lewis and Masters [377] and Ruskin and Decherd [528] have studied the effect of quinidine on auriculo-ventricular and intraventricular condition recovery. Conduction recovery curves for these areas before and after quinidine are similar to those shown for the auricle in Figure 295. Thus quinidine depresses conduction recovery in all three areas of the heart.

Observation 3: Effect of Quinidine on the Motion of the Fibrillating Auricle

Auricular fibrillation was produced in five dogs by the application of aconitine to a site on the surface of the right auricle. Cinematographs were made at 1,000 frames per second, simultaneously with electrocardiograms. Quinidine lactate was then given intravenously in fractional doses of 20 milligrams every 20 to 30 seconds and photographs taken after each dose. Thus progressive changes in the motion and appearance of the same auricle from the unquinidinized state to complete quinidinization (about 10 to 20 milligrams per kilogram) were observed.

After quinidine was injected, the contraction waves in the fibrillating auricle gradually became stronger and traveled greater distances, this effect frequently could be seen with the naked eye. Dilation of the auricle was observed (Figure 296). As additional quinidine was received, the motion throughout the contractile auricular musculature showed progressive decrease in rate accompanied by increasing coordination and organization until suddenly fibrillation terminated. Onset of either auricular flutter or a slower rhythm was marked by a disappearance of the minute areas of contraction and the spread of large contraction waves across the entire contractile auricular surface. The large contraction waves then recurred at regular and slower rates.

Observation 4: Effect of Quinidine on the Electrical Activity of the Fibrillating Auricle

Ten dogs were anesthetized and the heart

exposed. Auricular fibrillation was produced by application of aconitine to a site on the surface of the auricles Continuous direct auricular and limb lead electrocardiograms were taken while quinidine lactate was administered intravenously in an average dose of 20 mgm. per kilogram. In each instance the tracings showed a rapid change to normal sinus rhythm following the injection. The rhythm often changed from fibrillation to flutter, then to tachycardia and finally to sinus rhythm * In instances, intermediate arrhythmias were skipped and a direct change from fibrillation to sinus rhythm occurred.

The clinical counterpart of these events frequently is seen during treatment of human auricular fibrillation with quinidine.[370]

In eight dogs auricular fibrillation was produced by the application of aconitine to a site on the exposed right auricle while a direct auricular lead was recorded on the oscilloscopic screen and photographed with either the Fastax Camera or the Du Mont Oscillograph Record Camera Quinidine lactate was given intravenously in fractional doses as described in Observation 3 and oscillographic records were made at intervals. The oscillograms exhibited a marked change following the initial injection of quinidine. The large ("L") auricular deflections increased in amplitude, some becoming as large as the ventricular complexes, occurred less frequently and perhaps more regularly, and appeared more symmetrical; however, they were still irregular in rate and no two were identical. The minute ("M") waves seemed smaller. As additional quinidine was given, progressive slowing of the auricular rate occurred. The baseline between the large waves became smoother and suddenly fibrillation ceased with disappearance of the minute waves. Flutter then occurred in most instances, followed by tachycardia and normal sinus rhythm; the oscillographic deflections of these slower-rate rhythms were consistently symmetrical and regular in rate (Figure 297).

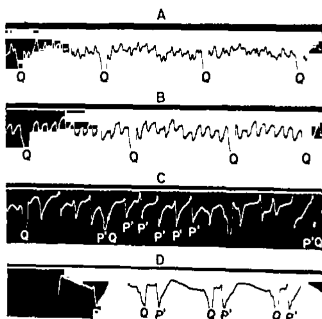


Figure 297. Direct auricular lead oscillogram from a dog exhibiting transitions from fibrillation to auricular tachycardia induced by quinidine. (Time markings 1/120 second.)
(A) Untreated fibrillation.
(B) Fibrillation immediately following quinidine The oscillations occur at a slightly slower rate than *in A and* bear some resemblance to one another.
(C) Intermediate stage in the transition from fibrillation to flutter. Note the increased amplitude and decreased rate of the deflections. Aberration of the P' waves is marked.
(D) Auricular tachycardia. The P'-Q interval is prolonged.

A similar experiment was performed while two direct auricular leads were recorded simultaneously on the oscilloscope. Each lead exhibited the changes described above. The large auricular deflections became more symmetrical and the deflections in the two leads became more synchronized with one another. As the animal received more and more quinidine, the synchronicity of the paired leads progressively increased. Complete synchronicity was achieved with the termination of fibrillation

Cinematographic Observation 3 may be correlated with electrocardiographic Observation 4. As shown cinematographically, contraction waves are conducted decrementally and unpre-

* A similar series of events was observed following administration of quinidine to five dogs exhibiting auricular flutter as an after-effect of electrical stimulation Since poststimulatory auricular arrhythmias usually are of short duration, however, it was often difficult to determine whether the conversion to sinus rhythm was spontaneous or due to quinidine

dictably for short distances in untreated fibrillation. As the rate of auricular activity is slowed by quinidine, the waves travel for increasing distances until eventually they spread across the entire surface of the auricle. At this stage, the minute ("M") activity of fibrillation is terminated and flutter or a slower-rate rhythm ensues. These cinematographic changes correspond to the oscillographic changes observed during the quinidine-induced transition from auricular fibrillation to normal sinus rhythm (Figure 297). The oscillogram exhibits progressive decreases in the frequency of the large auricular oscillations after administration of quinidine; as the oscillations occur at slower rates, they become progressively more organized. When sufficient slowing of the rate takes place, regularity and organization of the auricular deflection is achieved, fibrillation terminates, and a pattern characteristic of flutter is seen. The cinematographically observed decrease in decremental conduction after administration of quinidine correlates with the finding that quinidinization is followed by increased synchronicity and similarity between simultaneous oscillograms obtained from paired electrodes on the surface of the auricle.

The Effect of Quinidine on the Rate of Discharge from the Ectopic Focus

The above observations suggest that the primary anti-arrhythmic action of quinidine is a slowing of the rate of discharge from the ectopic focus (Observations 3 and 4). Cinematographic and electrographic changes identical with those induced by quinidine have been observed when the rate of discharge from an aconitine produced focus is slowed by cooling the focus. When the auricular rate is controlled, quinidine slows conduction and delays conduction recovery. When the auricular rate is not controlled, however, the quinidine-induced slowing of the rate permits more complete conduction recovery in rapidly beating auricles. At present the precise pharmacologic action responsible for the ability of quinidine to decrease the rate of discharge from the ectopic focus is obscure but changes in the threshold of excitability and in conductivity in the area of the focus undoubtedly are of major importance.*

The therapeutic effect of quinidine is summarized in Figure 298 by points W, X, Y and Z representing the progressive slowing in the rate of discharge from the ectopic focus which follows quinidinization during fibrillation. For purposes of illustration, Y may be considered the point at which slowing of the auricular rate by the drug results in sufficient organization of the auricular deflection to produce a recognizable "flutter wave." With further slowing of the auricular rate, the deflection would resemble the P′ wave of auricular tachycardia (point Z).**

As the rate of discharge from the ectopic focus decreases, the length of the cardiac cycle increases; the interval during which the auricle is permitted to recover from the previous excitation increases and conduction recovery becomes more complete. When the rate of discharge from the ectopic focus falls below the rate from the sino-auricular node which is relatively more resistant to quinidine, the normal pacemaker takes over and normal sinus rhythm then exists. Sudden changes in the rate of discharge from the ectopic focus result in the skipping of intermediate stages and a direct conversion from fibrillation to normal sinus rhythm.

The reasons for clinical failures of quinidine cannot be stated with certainty. Most such instances probably are attributable to one of two

* The effect of quinidine is to drastically delay recovery of auricular excitability and conductivity which follows the absolute refractory period. Inasmuch as the ectopic focus of an auricular arrhythmia exists in cardiac muscle, it seems reasonable that this effect may either delay impulse formation in or prevent the conduction of the impulses out of the ectopic focus. In either event, the rate at which impulses are discharged from the ectopic focus would be slowed. Sinus rhythm returns when the activity of the ectopic focus is affected to a greater degree than the sinus node which is normally more resistant to quinidine It is possible also that the effect of quinidine on the ectopic focus is partially due to its ability to decrease vagal tone[61] since vagal stimulation is known to favor the establishment of auricular fibrillation (Chapter 14).

** For the purposes of simplicity we have described these events as occurring in orderly sequence. Actually, they occur in a stair-step order, at times progressing and at times regressing

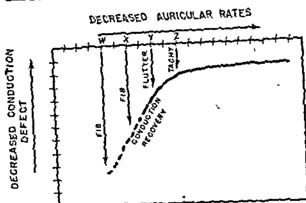


Figure 298. Hypothetical drawing summarizing the cinematographic and oscillographic events observed during the quinidine induced termination of auricular fibrillation. Arrows W, X, Y, and Z indicate progressive decreases in the rate of discharge from the ectopic focus The decrease in rate effected by quinidine permits a greater degree of conduction recovery to take place and thus permits the transition from fibrillation to a slower-rate arrhythmia This is the reverse of the events which lead to the production of auricular fibrillation (Figure 259, Chapter XIV)

factors: (1) inadequate blood levels (this factor has been emphasized recently by Sokalow,[372] (2) an ectopic focus whose resistance to quinidine equals or surpasses that of the *sinoauricular* node

Part II

THE PHARMACOLOGY OF DIGITALIS

As noted in Part I of this chapter, quinidine has a similar therapeutic effect on all the clinical and experimental auricular arrhythmias In contrast, the effect of digitalis on these arrhythmias is variable and at times seemingly unpredictable. Digitalis is anti-arrhythmic in auricular paroxysmal tachycardia and, with exceptions, pro-arrhythmic in flutter and fibrillation As in the case of quinidine, these effects have been ascribed by many investigators to alterations in the excitable gap of the hypothetical circus pathway, the more complicated action of digitalis, however, is even less adequately explained by the circus movement theory We have observed the effects of digitalis on the auricles and have correlated our observations with those of other workers in an attempt to achieve a better understanding of the mechanism involved

Clinical experience with the cardiac glycosides has been summarized recently by Kisch;[307] Movitt;[463] and Linenthal.[392, 393] Wollenburger[676] reviewed present-day knowledge concerning the effect of the cardiac glycosides on the metabolism of heart muscle, a subject less familiar to clinicians.

Observation 5: Effect of Digitalis on the Cinematographic Appearance of the Heart

In 10 dogs *cinematographs of* the heart during sinus rhythm were made with the Fastax Camera. Strophanthin K or acetyl strophanthidin (average dose 0.03 mgm. per kilogram) was then administered intravenously; when digitalis effects were manifest on the electrocardiogram, the heart was again photographed. The pre- and post-digitalization films were developed and projected simultaneously and adjacent to one another as in the study of quinidine. Thus one could compare directly, in the same *heart,* minute differences resulting from administration of the drug. Following digitalization the auricles were greatly decreased in size and their motion restricted. The ventricles also were decreased in *size.* Because of these changes, the rate of spread of the contraction wave in the auricle could not be estimated.

Similar pre- and post-digitalization cinematographs of the right auricle were recorded during aconitine-induced auricular fibrillation in eight dogs. The same observations also were made in four dogs during electrical stimulation of the right auricle at various rates Comparison of the films revealed that after digitalization the *fibrillating auricle* became smaller, appearing as a contracted mass of quivering muscle (Figure 299).

A comparison of the above observation with Observation 3 (Figure 296) demonstrates that digitalis and quinidine have opposite effects on the size of the heart. This difference is particularly notable *in the* auricle.

Clinical Correlation: The salutory effect of digitalis in heart failure is well known and is generally believed to result from the increased

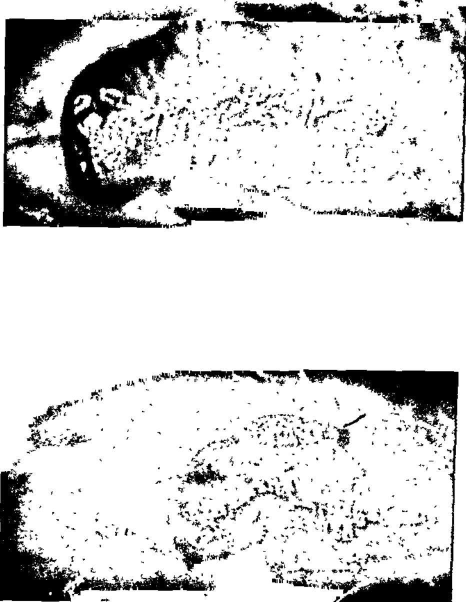


Figure 299 Stills from motion picture to demonstrate the effect of digitalis on the auricular myocardium Upper photograph shows maximal diastole of auricular fibrillation produced by aconitine in a dog Lower photograph shows maximum diastole after digitalis was administered The auricle continues to fibrillate Note that the auricle is much smaller in size after digitalis has been administered, possibly due to increase in myocardial tone. The effect of digitalis is opposite to that of quinidine as demonstrated in Figure 296.

force of contraction of heart muscle under the influence of the drug (cardiotonic action). The effect is readily demonstrable by mechanical methods[95, 646] and has been visualized in high-speed cinematographs of experimental heart failure produced by the intravenous infusion of saline solution. Under these experimental conditions, the administration of strophanthin was followed by a return of the heart size *toward* normal; other changes included an increase in the amplitude of the contraction waves, an increase in the degree of systolic emptying, and a lengthening of the interval occupied by diastole, thus permitting greater diastolic filling. All these factors and possibly others[408, 450] tend to increase cardiac output in heart failure associated with a decreased cardiac output. As noted in the above experiment, when strophanthin was administered to animals without heart failure, the heart size decreased to *less* than normal. This is offered as a possible explanation for the well known fact that digitalis often decreases the output of a normal heart

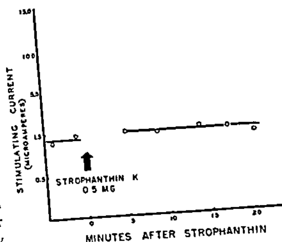


Figure 300. Threshold of auricular excitability determined for an auricular rate of 200 before and after digitalization The threshold current required to drive the auricle at a rate of 200 is not significantly changed by non-toxic doses of strophanthin However, the current required to drive the auricles at extremely rapid rates after digitalization is greater than that required in the unmedicated animal Anesthesia, pentobarbital.

The Effect of Digitalis on Vagal Tone

It is a well established fact that vagal stimulation results from the administration of drugs belonging to the digitalis group At least part of this increase in vagal tone has been found to arise reflexly in the carotid sinus[279] Since increased vagal tone is an integral part of digitalis action, changes in cardiac rhythm due to vagal stimulation represent *indirect* effects of digitalis These indirect effects are considered concurrently with the direct effects of digitalis in the following paragraphs.

The Effect of Digitalis on the Absolute Refractory Period

As in early studies of quinidine, workers prior to 1925, with the notable exception of Clark and Mines,[97] reported that digitalis lengthens the absolute refractory period of heart muscle Love,[394] however, demonstrated that when measures are not taken to eliminate the error introduced by incomplete conduction recovery, the values obtained for the absolute refractory period are too high. Working under conditions eliminating or decreasing this error, Love;[394] Macleod;[421] and Wedd, Blair and Dwyer[620] found that digitalis actually shortens the absolute refractory period.

The Effect of Digitalis on Auricular Excitability

We have confirmed observations of other investigators[620] that the threshold current required to drive the auricles at relatively slow rates is not significantly altered by non-toxic doses of digitalis (Figure 300). To drive the auricles at extremely rapid rates, a significantly stronger current is required following digitalization[183] Toxic doses of digitalis, like non-toxic doses of quinidine, raise the excitability threshold of all auricular rates

To our knowledge, the effect of increased vagal tone, or the indirect effect of digitalis, on the threshold of auricular excitability has not been studied for all auricular rates. Increased vagal tone has been found to increase excitability in mammalian auricles and to de-

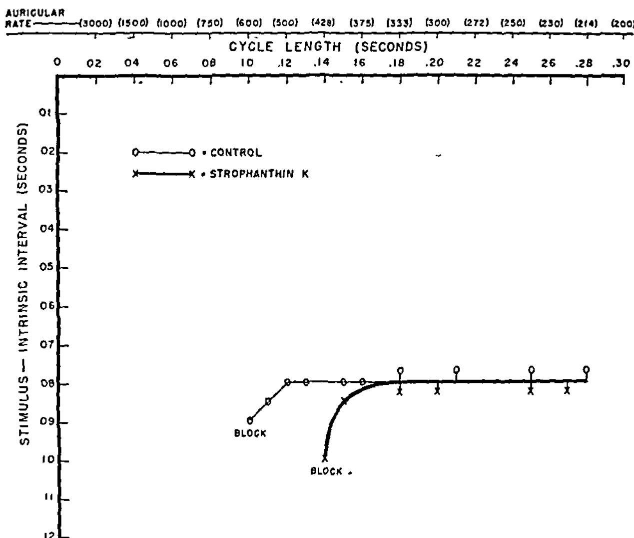


Figure 301. Conduction recovery before and after digitalization. Following administration of strophanthin K (0 06 mgm/Kg) conductivity is unchanged at slower rates of stimulation. Comparison of the two curves indicates that conduction recovery is delayed by strophanthin Anesthesia, Urethane.

crease excitability in the auricles of cold blooded animals.[518] It is possible that factors of auricular rate account for this apparent difference in effect of vagal tone on excitability.

Observation 6. The Effect of Digitalis on Auricular Conduction Recovery

In 18 dogs with heart exposed, direct auricular leads were simultaneously recorded from the body of the right auricle and the left auricular appendix. A stimulating electrode from a low frequency pulse generator was attached to the right auricular appendix, and the positions of all electrodes were left unchanged for the duration of the experiment. The auricle was then stimulated with currents several times threshold at rates in gradual increments of 25-50 stimuli per minute while continuous electrocardiograms were recorded at double speed (50 millimeters per second). Such records were made before and after the administration of fractional doses of strophanthin K at intervals of 20 minutes; the total dose of strophanthin seldom exceeding 0.05 mgm. per kilogram of body weight. Measurements of the stimulus-intrinsic interval were made as in Observation 2. As in the case of quinidine, when the auricles were stimulated at increasing rates following the administration of strophanthin K, manifestations of conduction failure occurred at slower auricular rates than in the unmedicated animal (Figure 301). At

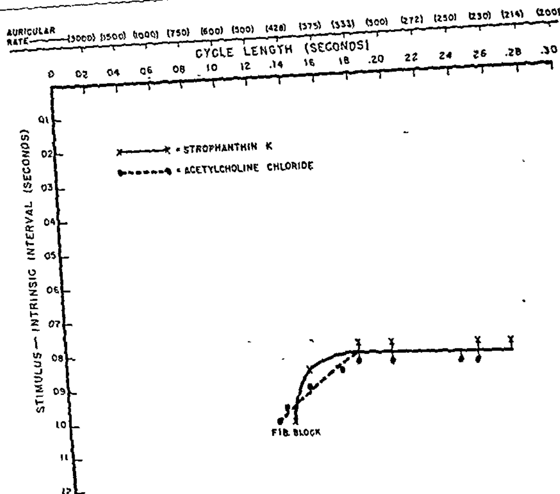


Figure 302 Conduction recovery in the digitalized animal determined during the administration of a constant acetylcholine drip. Note that the gradient of conduction recovery after digitalization is altered by the administration of acetylcholine, thereby making it possible to produce fibrillation. The antagonism between the effects of strophanthin and acetylcholine on conduction recovery is clearly demonstrated

slow auricular rates, however, conduction recovery following digitalization was complete. When digitalis was administered in toxic doses, incomplete conduction recovery was observed at all auricular rates. Thus the effect on conduction recovery of toxic doses of digitalis is similar to that of non-toxic doses of quinidine (Observation 2). Figure 301 shows the effect of strophanthin on auricular conduction recovery in an auricle which blocked both before and after the administration of the drug Figure 302 shows the same auricle after administration of strophanthin and with further alteration of conduction recovery produced by the slow intravenous administration of acetylcholine. These and similar observations suggest a relationship between the gradient of the conduction recovery curve and the ability to produce auricular fibrillation by electrical stimulation.* Further investigation of this problem would seem desirable.

As in the case of quinidine, the occurrence of auricular blocking at slower rates of *stimulation* following digitalization may be due to conduction failure.[394]

Conduction recovery curves drawn for the

* Under uniform conditions, the gradient of conduction recovery is probably inversely proportional to changes in the threshold of excitability which occur during the terminal portion of the relative refractory period (See also Chapter XIV)

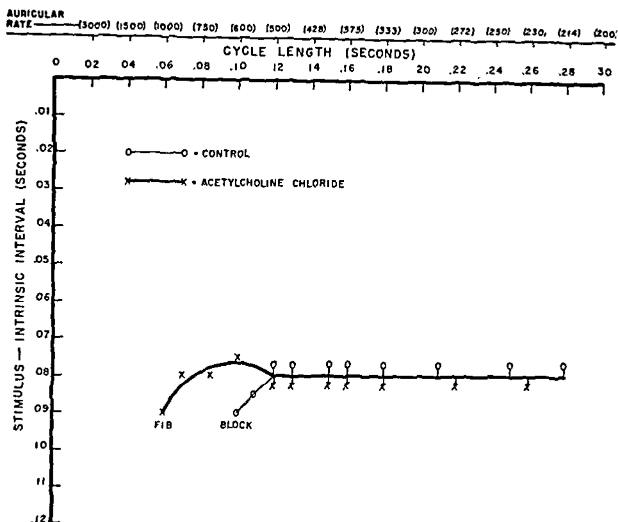


Figure 303. Auricular conduction recovery before and during the controlled administration of acetylcholine. *The curve indicates that acetylcholine not only accelerated conduction recovery but also induced a supernormal phase* of conduction recovery between the auricular rates of 500 and 750 per minute *In some experiments this acetylcholine induced supernormal phase was observed at auricular rates as low as 350 per minute (see also Figure 302)* (Anesthesia, Urethane.)

ventricles[136, 528] and the auriculo-ventricular conducting system[377, 528] before and after digitalis resemble those shown for the auricles (Figure 301). Thus digitalis delays conduction recovery in all three areas of the heart.

The effect of acetylcholine, or the indirect effect of digitalis, on auricular conduction recovery was studied in several animals. An intravenous drip of an acetylcholine solution* was administered at a constant rate just fast enough to produce sinus bradycardia. The auricles were then driven electrically and changes in conduction recovery determined in the manner described in Observation 2 Auricular conduction recovery was greatly facilitated by acetylcholine. Figure 303 represents an instance in which conduction acceleration occurred at auricular rates of 600, this is regarded as a phase of supernormal conduction recovery and is more clearly demonstrated in Figure 262A. *Rosenbleuth and Garcia Ramos*[518] have reported similar auricular conduction acceleration at rapid auricular rates following vagal stimulation.

The vagus has long been recognized to facilitate intra-auricular conduction, especially when intra-auricular block is present.[151, 152, 376] No completely satisfactory explanation for these phenomena has been advanced. The effect of vagal stimulation at the auriculo-ventricular node apparently is quite different than in the

* Acetylcholine chloride 100 mgm. dissolved in 200 cc. of distilled water.



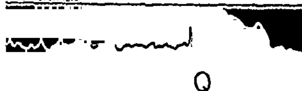


Figure 304 Direct auricular lead oscillogram from dog showing auricular fibrillation before and after digitalization Following digitalization the oscillations are of decreased amplitude and appear to occur at a more rapid rate (Time marking 1/120 second.)

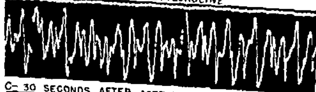
auricles. Under the influence of vagal stimulation, conduction recovery at the auriculoventricular node is delayed[377] and conduction in this area fails at relatively slow auricular rates Vagal stimulation appears to have no effect on intraventricular conduction,[151] although the ventricular myocardium is apparently susceptible to the action of cholinergic drugs

Observation 7. Effect of Strophanthin on the Electrogram from the Fibrillating Auricle

In 18 dogs, auricular fibrillation was produced by the application of aconitine to a point on the surface of the right auricle and a direct auricular lead was recorded from the oscillograph with the Fairchild camera. Fractional doses of strophanthin or acetyl strophanthidin were administered intravenously every twenty minutes and intermittent oscillographic records were made Examination of the records thus obtained usually revealed a progressive decline in the amplitude of the auricular deflections, with a resultant quieting and smoothing of the baseline between ventricular complexes (Figure 304) This event took place before toxic doses of these drugs had been administered A similar effect was observed in animals in which both vagi previously had been sectioned high in the neck, but in these instances the electrocardiographic changes were not studied quantitatively. Several animals in which auricular fibrillation had been produced were given acetylcholine chloride (0.5 to 1.0 mgm.) before and after digitalization while continuous records were made with the oscillograph. In these instances the rate and amplitude of the auricular deflections increased (Figures 305 and 306).

A clinical counterpart of the above observation was made in three patients with auricular fibrillation by recording esophageal lead oscillograms before and after digitalization. Following digitalization, the amplitude of the deflections recorded from the level of the fibrillating





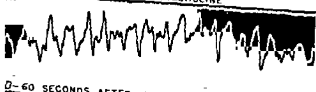




Figure 305 Direct auricular lead ...

... seconds after administration of acetylcholine. Note the increased frequency of the electrical oscillations. (C) Thirty seconds after administration ... line The ... (D) S ... etylcholine Tracin ... etylcho- ... almost returned to pre-treatment state.

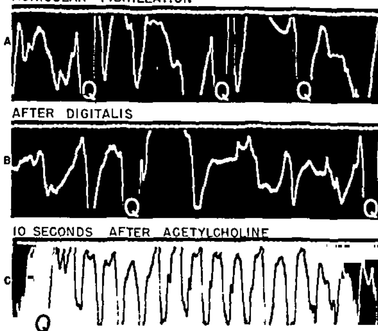


Figure 306. Direct auricular lead oscillogram from a dog showing the effect of acetylcholine in auricular fibrillation already treated with digitalis. (Time marking 1/120 second.)

(A) Untreated auricular fibrillation.

(B) After administration of digitalis. The amplitude of the oscillations is reduced.

(C) Ten seconds following acetylcholine administration. Note the opposite effects of acetylcholine and digitalis on the amplitude of the auricular deflections

auricles was greatly diminished and there was relative calm between the ventricular complexes (Figure 307). Similar observations of the effect of digitalis on clinical auricular fibrillation were made by Hart [262] using the ordinary string galvanometer.

It seems reasonable to attribute the effect of digitalis on the oscillogram from the fibrillating auricle at least partially to a further increase in the already marked conduction defect *present in the arrhythmia. The diminution in* electrical "bombardment" of the auriculo-ventricular node which apparently occurs following digitalization (Figures 304 and 307) may be one of the factors contributing to the slowing of the ventricular rate observed when auricular fibrillation is treated with digitalis.

The Effect of Digitalis on Auricular Conductivity

The *direct* effect of digitalis is to delay conduction recovery and thereby decrease conductivity in all three areas of the heart. The effect of increased vagal tone, or the *indirect* effect of digitalis, is to accelerate conduction recovery and increase conductivity in the auricles. Increases in vagal tone delay conduction recovery and thereby reduce conductivity of the auriculo-ventricular conducting system, but have no effect on the ventricles. In the auricles the direct and indirect effects of digitalis are antagonistic; however, in our experiments, the net effect of the drug in this area usually delayed conduction recovery. In the auriculo-ventricular conducting system, the direct and indirect effects of digitalis are complementary [234] and conductivity between the auricles and ventricles is greatly reduced by the drug. In the ventricles, only the direct effect of digitalis is operative and conductivity is decreased.

Both digitalis and quinidine delay conduction recovery and decrease conductivity in all areas of the heart. Quinidine, in contrast to digitalis, decreases vagal tone. In the auricles, quinidine depresses conduction more drastically than comparable doses of digitalis; unlike digitalis, quinidine produces a state of incomplete *(or infinitely delayed) auricular conduction* recovery. In the ventricles, where changes in vagal tone do not affect conductivity, quinidine also appears more active than digitalis in delaying conduction recovery [136, 528] Although quinidine apparently decreases conductivity in

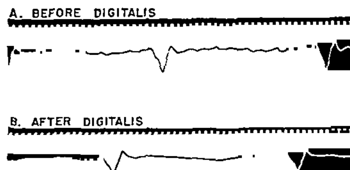


Figure 307. Esophageal lead oscillograms from patient before and after digitalis Following digitalization the undulations of the base line between ventricular complexes almost disappear (Time marking 1/120 second. Low amplification was used in this recording but standardization of the two tracings is identical)

the auriculo-ventricular conducting system, its depressant action in this area is partially counteracted by the decrease in vagal tone and is less striking than that of digitalis whose direct and indirect effects on auriculo-ventricular conduction are complementary.

The Effect of Digitalis on the Rate of Discharge from the Ectopic Focus

In both clinical and experimentally produced auricular arrhythmias, digitalis and vagal stimulation usually have identical effects on auricular rate.[188, 189, 370, 371, 375, 392, 393, 549] When the rate of discharge from the ectopic focus is rapid, both digitalis and vagal stimulation tend to increase the rate. When the rate of discharge is slow, both measures tend to decrease the rate.* Thus the complicated pro- and anti-arrhythmic action of digitalis is determined by the ability of the drug to increase vagal tone.

Lewis and associates[370, 371] apparently were the first to appreciate the importance of auricular rate in determining the response to vagal stimulation These investigators found that if the auricles were beating around 400 per minute, vagal stimulation usually caused an automatic acceleration of the rate and the production of auricular fibrillation. At slower auricular rates, vagal stimulation more often caused abrupt cessation of the arrhythmia or had no effect at all. Similar results were obtained by Scherf and associates[549] in a study of aconitine-induced arrhythmia. We have confirmed these observations by administering acetylcholine while the auricles were beating at various rates in response to electrical stimulation or during aconitine induced arrhythmias (Chapter 9) (Figures 194 and 195). Although it is impossible to determine the auricular rate when fibrillation exists, an increase in vagal tone during this arrhythmia has long been known to result in an increased number of oscillations in the electrocardiogram. This suggests that vagal stimulation during fibrillation also accelerates the rate of auricular activity.

At present the question must be regarded as incompletely answered why vagal stimulation may result in asystole when the auricles are beating relatively slowly, and yet accelerate an already rapid auricular rate.

SUMMARY AND CONCLUSION

Quinidine depresses both auricular excitability and auricular conductivity for any given auricular rate. In auricular fibrillation and rapid flutter, quinidine permits more complete conduction recovery in rapidly beating auricles by slowing the rate of discharge from the ectopic focus.

Both experimentally and clinically, quinidine is effective in terminating all the auricular arrhythmias. This anti-arrhythmic action is related to the ability of the drug to slow the rate of discharge from the ectopic focus. The termination of an auricular arrhythmia by quinidine may be either gradual or abrupt. During the quinidine induced conversion of auricular fibrillation to normal sinus rhythm, intermediate stages of flutter, auricular paroxysmal tachycardia and auricular premature systole often appear as the rate of discharge from the ectopic focus is gradually slowed by the drug.

The effect of digitalis on the auricles con-

* As suggested in a footnote [illegible] quinidine induced [illegible] conduction recovery [illegible] at which impulses a[illegible] ectopic focus, thus [illegible] to decrease the rate of discharge from the ectopic focus during auricular arrhythmias The direct effect of digitalis likewise is to depress auricular excitability and conduction recovery, although to a lesser degree than quinidine. This may account for the observation that digitalis slightly slowed the rate of discharge from the ectopic focus during auricular flutter in vagectomized dogs.[?] The suggestion that changes in the rate of discharge from an ectopic focus on the auricles may be due to changes in the rate at which impulses are formed in and/or conducted from the focus, and that these changes at the focus in turn are brought about by changes in auricular excitability and conduction recovery, may also be [illegible] tion on the auricle. [illegible] lar excitability and [illegible] induce a supernor[illegible] the auricles are be[illegible] of [illegible] hypothetical sequence of events [illegible] from the focus. Such a fact that vagal stimulation increases the auricular rate when the auricles are beating rapidly, but fails to explain why the same procedure slows the auricular rate during slower-rate arrhythmias

sists of (1) a direct action resembling but quantitatively less than that of quinidine; and (2) an indirect action, exerted through an increase in vagal tone, which tends principally to affect auricular rate but also has a complicated effect on auricular conductivity. Both these actions are manifest in the auricular arrhythmias but the indirect action, or the effect of vagal tone, determines the complicated pro- and anti-arrhythmic effect of the drug. Nontoxic doses of digitalis decrease both conductivity and excitability in rapidly beating auricles but fail to affect these factors when the auricle is beating at slow rates.

In both the experimentally produced and clinical auricular arrhythmias, the electrocardiographic events following digitalis administration are indistinguishable from those following vagal stimulation or the administration of a cholinergic drug. The net effect of digitalis in a given arrhythmia depends principally upon two factors: (1) the auricular rate; and (2) the degree of increased vagal tone produced by the drug. The duration of the arrhythmia and the presence or absence of heart failure are also of clinical importance but their role in determining the effect of digitalis is poorly understood.

Auricular paroxysmal tachycardia, generally a slow arrhythmia, frequently is terminated by digitalis. The termination of auricular paroxysmal tachycardia is usually electrocardiographically identical whether achieved by administration of digitalis, by the administration of a cholinergic drug, or by carotid sinus stimulation. In all three instances a variable period of asystole occurs following which sinus rhythm is re-established.

In flutter, generally a rapid arrhythmia, the administration of digitalis may produce an acceleration of the auricular rate culminating in fibrillation. In fibrillation, administration of digitalis usually perpetuates the arrhythmia and appears to increase the rate of auricular activity. The same sequence of events may take place following the administration of a cholinergic drug or vagal stimulation under similar conditions.

The events leading to the termination of auricular fibrillation by digitalis remain obscure. Such conversion occasionally is seen clinically; in these instances the fibrillation often is of the acute or paroxysmal rather than the chronic type. Some instances in which fibrillation is terminated by digitalis may be attributed to an improvement of existing heart failure.

The theory that anti-arrhythmic drugs terminate auricular arrhythmias by abolishing an excitable gap on a circus pathway is erroneous. Although much remains to be learned concerning the pharmacology of quinidine and digitalis, the effect of these drugs on the auricular arrhythmias can be explained without resorting to the circus movement theory

CHAPTER XVII

Treatment of the Auricular Arrhythmias

THE TREATMENT of auricular arrhythmias requires astute judgment in addition to sound scientific knowledge Rarely in clinical practice is the combination of science and art more necessary Although experienced physicians generally treat the auricular arrhythmias properly, the young doctor learns only after long, painful, and sometimes tragic experience.

In the majority of textbooks and teaching institutions the therapy of auricular arrhythmias is described as the treatment of a disease *per se*. Such instruction is misleading, for the successful application of therapeutic measures depends upon proper consideration of three factors· (1) the general condition of the patient, such as age, shock, etc , (2) the condition of the myocardium, including presence or absence of myocardial failure, coronary disease, irritable ventricles, (3) the physiologic disturbance in the auricles. Here, the electrocardiogram with auricular leads is often helpful if properly interpreted. In a severe case, all three of these factors must be thoroughly considered. Auricular paroxysmal tachycardia with a rate of 200 per minute in a 20 year old man should be treated quite differently from a paroxysmal tachycardia in a man aged 70 in peripheral shock with myocardial disease. Yet, the electrocardiograms may not be unlike in the two instances. The fact that these general principles of therapy are not well appreciated is illustrated by the frequency with which intelligent, well-informed physicians ask us to recommend the best treatment for a certain auricular arrhythmia, or inquire whether or not we believe that fibrillation should be converted to sinus rhythm.

The arrhythmias frequently have a dramatic impact on the inexperienced physician which may cause him to try one remedy after another without regard to the three factors mentioned above. After a brief preliminary consideration of these factors, he generally can proceed according to the rule that the simpler therapeutic measures should be applied first. Since the patient and family are apprehensive, sedation usually is advisable. Morphine is an excellent sedative and often induces vomiting which often terminates the arrhythmia.

Fortunately, many therapeutic measures are available and the response to treatment is often as dramatic as the clinical picture is alarming. The physician and student should be thoroughly familiar with the principles and methods of application of a few basic procedures. With the present state of knowledge, carotid sinus massage, parenteral and oral use of digitalis, parenteral and oral use of quinidine are essentials in the armamentarium of the practicing physician. These measures will enable him to treat successfully all but the rarest auricular arrhythmias. He should not try new anti-arrhythmic drugs as soon as they appear, but rather should rely on proven therapies until the new procedures become established. Premature clinical application of new drugs may prove fatal, as exemplified by the tragic effects of fagarine in certain instances. The physician who can effectively apply established measures will be able to keep an open but critical mind concerning the new ones.

Among the many anti-arrhythmic measures available are various mechanical procedures

and drugs which exert their effect through vagal stimulation, and a number of drugs which have a direct action on the auricular myocardium with or without additional vagal effect. Some of these procedures and agents are effective in the treatment of one or two of the arrhythmias, others are useful in all. A brief description of the more important anti-arrhythmic measures and their methods of application follows.

VAGUS-STIMULATING MECHANICAL PROCEDURES

Carotid Sinus Massage: [100] In our experience, this procedure when properly utilized is the most effective mechanical method of exciting vagal action. Pressure over the carotid artery is often recommended, but the correct procedure is *massage* of the carotid sinus. Physicians not infrequently report that they have found carotid sinus pressure ineffective, when actually the only result of their maneuver was to obliterate the carotid artery below the sinus.

Carotid sinus massage yields the best results in those patients with long, narrow necks. The patient should be in the supine position, this is in contrast to the sitting or standing position recommended when carotid sinus stimulation is used to determine the cause for syncope. The head is comfortably extended with a pillow under the neck and is turned slightly to one side. A sedative will help assure that the patient is not tense and that the platysma muscle is relaxed. After the carotid sinus is located as a bulge on the artery, the thumb is used to administer slow but firm strokes up and down the artery for a distance of 2 to 4 cm Massaging should be done first on the right side and then on the left; both sides should never be stimulated simultaneously. The following technique is most efficacious: the physician stands on the patient's right side, allowing the patient to hold the bell of the stethescope at the heart's apex with his left hand while the physician listens to the apex and massages simultaneously. As the massage is applied, the patient slowly counts aloud to 20. This device helps distract the patient from the annoyance of the procedure and provides a maximum time limit for uninterrupted massage; if the carotid sinus is hypersensitive (type III, Weiss [625]) and the patient faints, the physician is notified as the counting ceases. If the arrhythmia is interrupted during the procedure, massage is stopped immediately. This procedure taught by the physician can be used by the patient himself or by some member of his family in those instances in which the arrhythmia recurs frequently. The beneficial results of carotid sinus massage in auricular arrhythmias, in our experience, have been directly related to the skill with which the physician utilizes the procedure.

Askey [8] has directed attention to deleterious results from carotid sinus stimulation. Severe reactions such as transient or permanent hemiplegia or death due to thrombosis of bilateral anterior cerebral arteries [431] have been recorded. These occurrences, however, were noted only in elderly arteriosclerotic patients with the hypersensitive carotid sinus syndrome. Such instances are not due to cerebral ischemia resulting from occlusion of the carotid artery, nor are they necessarily due to reflex changes in blood pressure or asystole; as shown by Weiss,[625] the untoward reactions probably represent a reflex cerebral effect. Thus carotid stimulation should be used with caution in individuals, especially elderly patients, who faint frequently. Also it is apparent that the procedure can safely be used in younger patients in whom it is most effective therapeutically.

Ocular Pressure: The physician who can do carotid sinus massage properly will find ocular pressure an obsolete and seldom useful method of treating arrhythmias. Ocular pressure induces vagal stimulation by mediating the stimulus through the ophthalmic fibers of the trigeminal nerve Since the procedure is somewhat painful, an analgesic may be given prior to its application. Ocular pressure is most valuable in aged sclerotic patients in whom it may be used to avoid the danger of unfavorable side effects associated with carotid sinus massage.

Also it is of value in patients with short, thick necks in whom proper carotid sinus massage is difficult. To apply ocular pressure, the physician should have the patient supine, with his eyes closed and his gaze directed downward. Pressure is applied on the superior surface of the eyeball above the level of the cornea, and with sufficient firmness to cause some pain. Pressure should not be maintained for longer than 20 seconds at a time, and the physician should be listening at the apex throughout the procedure. Again, the patient should be instructed to count aloud to 20. In our experience, ocular pressure is inferior to carotid sinus massage as a means of stimulating the vagus. It is unlikely that ocular pressure will be successful in terminating an arrhythmia after proper carotid sinus massage has failed, although such occurrences have been reported.

Induction of Vomiting: During the act of vomiting, efferent impulses are discharged over many parasympathetic pathways including the vagal tract Vomiting may be induced by mechanical stimulation of the pharynx or by administration of ipecac.[626] Eight cubic centimeters of the syrup (U S P) is administered as an initial dose and repeated three or four times at hourly intervals if reversion to sinus rhythm is not obtained earlier. The amount of the drug required to obtain results varies widely in different patients, for a given patient, however, when the effective dose is once determined the same amount of the drug is apt to prove successful in terminating subsequent attacks Apomorphine,[194] although effective in the production of vomiting, is not recommended because of its toxic effect on the central nervous system. Vomiting as a method of vagal stimulation is not, in our experience, a practical therapeutic procedure.

Valsalva and Allied Procedures: These procedures generally are not successful in terminating auricular arrh[illegible] are frequent. The patient is instructed to exhale forcibly while the mouth is closed and the nose held by thumb and forefinger, or to exhale forcibly against a closed glottis Afferent impulses are sent to the vagus from the naso-pharyngeal mucosa through the fifth, seventh and ninth nerves. Vagal stimulation may also be obtained by deep breathing, holding the breath, or irritation of nasal mucosa.

Postural Changes and Miscellaneous Procedures: Other frequently recommended procedures include postural changes such as bending forward, stooping with head low and lying down with the head low, drawing out of tongue, swallowing of large bolus, pressing on abdomen, drinking of ice water, and placing of ice bag over precordium. We have found these "handed down" measures of no practical therapeutic value and it is inconceivable that they would be effective after proper carotid sinus massage had failed.

VAGUS-STIMULATING DRUGS

Available drugs which effect vagal activity either directly or indirectly include Mecholyl, Neostigmine, and Neosynephrine. These agents need be seldom employed to treat arrhythmias by physicians who can properly use quinidine and digitalis. Mecholyl occasionally may be indicated. Neostigmine and Neosynephrine have little or no place in the therapy of the auricular arrhythmias and are mentioned here only for the purpose of completeness.

Acetyl-Beta-Methylcholine (Mecholyl): Certain precautions must be observed when this powerful vagal stimulant is administered. The drug should be given subcutaneously only, *never intravenously*. A tourniquet should be tied loosely around the arm, and the injection made distal to it. As soon as the arrhythmia is terminated or toxic effects are obtained, the tourniquet is tightened and the action of the drug terminated since its effect is fleeting. The patient should be in a recumbent position to minimize cerebral effects, and, as Starr pointed out, it is practical to have the patient on a bedpan.[580] Atropine sulfate 1.0 mgm should be available in a syringe for immediate intravenous injection in the opposite arm and a vein suitable for injection should be selected; this pre-

caution is necessary to block the action of acetyl-beta-methylcholine in the event of the development of untoward side effects such as pronounced fall in blood pressure, nausea, vomiting, substernal pain, syncope or convulsions. The side effects of Mecholyl are often violent and the patient may not be pleased even if the arrhythmia is terminated. We have seen two instances of temporary and alarming asystole following subcutaneous administration of mecholyl.

Nathanson[466] recently described a method by which Mecholyl can be administered intranasally with an applicator. Advantages of the method include simplicity of administration, the fact that no sterile solutions need be used, and that the intensity of the reaction is governed by the length of time the applicator is in place. The suggestion was made that intelligent patients whose attacks are frequent and disabling might be taught self-administration of Mecholyl by this method. It is preferable to teach the patient or some member of the family the technique of carotid sinus massage because of the lesser hazard involved.

The average initial dose of acetyl-beta-methylcholine is 20 mgm. In youthful individuals who are more susceptible, 10 or 15 mgm. may suffice. Elderly, obese persons may require 30 to 60 mgm. Since the effect of the drug persists no longer than 15 or 20 minutes, a second and larger dose may be administered if the arrhythmia is still present after this period.

Mecholyl should not be given to allergic individuals, especially those subject to bronchial asthma. It should also be avoided in angina pectoris, in myocardial infarction whether acute or healed, and in hyperthyroidism. In patients with the latter condition, the drug may induce auricular fibrillation.

Neostigmine Methyl-Sulfate: Since this drug acts by inhibiting cholinesterase and allowing accumulation of choline in the body, its favorable and unfavorable effects are similar to those of acetyl-beta-methylcholine but less pronounced. Although some authors have reported[484, 612] Neostigmine effective in terminating auricular arrhythmias in small series of cases, its value as an anti-arrhythmic drug has never been established and remains questionable.

The average initial dose of Neostigmine is 0.5 to 1.0 mgm. intramuscularly. Maximum effects are obtained within 20 minutes. Immediate results may be obtained from intravenous administration but this method is apt to produce severe side effects. We have had no experience with this drug

Neosynephrine:[653] The therapeutic effect of Neosynephrine is attributed to reflex cardiac inhibition elicited by the rapid rise in pressure in the carotid sinuses and aortic arch. A diagnosis of supraventricular tachycardia must be established with certainty before neosynephrine is employed. The drug should never be used in ventricular tachycardia, coronary artery disease or hypertension. Side effects are minimal or absent; occasionally a tingling sensation of the skin or slight headache may occur.

The average initial dose of Neosynephrine in an adult is 1.0 mgm. given intravenously. In children and in lightweight persons, smaller doses may be used The drug is injected rapidly into the ante-cubetal fossa of one arm while the blood pressure is recorded at 30 second intervals from the other arm. When the therapy is successful, normal rhythm is established within 60 seconds. Unless blood pressure promptly rises 20 to 30 mm. Hg. or more, however, conversion will not be achieved. Repeated injections may be given at 10 minute intervals, increasing each successive dose by increments of 0.25 mgm. The value of Neosynephrine as an anti-arrhythmic agent has not been clearly established. Although the drug may terminate arrhythmias on occasion, we believe it inferior to the simpler and more direct methods of vagal stimulation.

DRUGS WHICH ACT DIRECTLY ON THE MYOCARDIUM WITH OR WITHOUT ADDITIONAL VAGAL EFFECT

Digitalis: Digitalis preparations, particularly the glycosides, are among the most useful

agents in the management of the auricular arrhythmias. This is especially true when the arrhythmia is associated with congestive failure; often restoration of compensation alone will establish normal sinus rhythm. Aside from its role in restoring myocardial efficiency, digitalis abolishes certain auricular arrhythmias by stimulating the vagus (Chapter XVI). The possibility that the degree of vagal action exhibited by certain glycosides differs from that of digitalis leaf is suggested by the work of Fahr[188] on lanatoside C. This investigator reported that in some of his patients without failure but with various auricular arrhythmias (chronic fibrillation, flutter, tachycardia) who had failed to respond to treatment with digitalis leaf exhibited a reversion to normal sinus rhythm following administration of lanatoside C. Other reporters[463, 597,598] believe that lanatoside C is the digitalis preparation of choice in the therapy of the auricular arrhythmias.

For the management of an acute attack, it is advisable to use a rapidly acting preparation (lanatoside C or ouabain, etc.) intravenously or intramuscularly. If the patient has received no digitalis for a period of at least two weeks, one-half of the expected digitalizing doses may be given at once (0.6 mgm. to 0.8 mgm. of lanatoside C or 0.5 mgm. ouabain); if the patient has been on "maintenance" doses of digitalis, a fraction of the digitalizing dose is slowly administered. Needless to say, one must first establish that the arrhythmia is not a manifestation of digitalis toxicity. The intravenous or intramuscular route may be used. In either case subsequent fractional doses are given at two- to four-hour intervals until effective digitalization (short of toxic effects) has been accomplished. If complete digitalization fails to restore normal sinus rhythm, carotid sinus massage should be tried followed by other procedures which increase vagal tone. After the arrhythmia has been terminated, maintenance doses of digitalis are given orally to prevent recurrence.

In most instances the maintenance dose of digitalis is one or two tablets of whatever preparation is being used. One of the disadvantages of giving digitalis in tablet instead of tincture form is that physicians fail to advise fractional doses. In some instances of fibrillation, especially in senile patients, we have found fractional amounts of tablets adequate. In these patients, the ventricular rate is an excellent guide to effective digitalization. When congestive failure is present, underdigitalization frequently occurs due to strict adherence to dosages supplied in tablets.[493]

Quinidine:[139, 235, 297, 298, 299, 313, 409, 563, 622, 637, 672] This drug is widely used in all the arrhythmias. The fact that quinidine can be given with relative safety in either auricular or ventricular arrhythmias is of great advantage when the physician is in doubt as to the proper diagnosis. Were it not for certain detrimental side-effects, quinidine might be considered the ideal anti-arrhythmic drug since it raises the excitability threshold of an ectopic focus and is successful in terminating all four auricular arrhythmias. The physician should be cognizant of the fact that quinidine has an atropine effect; therefore, there is no need to try vagus-stimulating methods after quinidine is given. Digitalis and quinidine, however, may safely be given together. In fact, in some instances, quinidine is best given after or simultaneously with digitalis.

The hopes expressed for quinidine 15 years ago have not been entirely fulfilled, and it is entirely likely that a new drug will be found having the same or greater therapeutic effects, but without unfavorable side effects. At the present time, however, quinidine is the most valuable anti-arrhythmic drug available. The excellent review by Di Palma[143] on chemical structure suggests that the new drug may be a methoxy derivative of benzene or a substituted ammonium compound.

Many clinicians consider quinidine the best therapy in auricular paroxysmal tachycardia, but our experience indicates that carotid sinus massage or digitalis are more valuable. In the presence of an impaired myocardium, quinidine is definitely inferior to digitalis. On the other hand, quinidine is preferable to digitalis when

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Mild toxic effects such as tinnitus and moderate gastrointestinal irritation are not contraindications to continuance of the medication. However, severe gastrointestinal irritation (vomiting, diarrhea), a marked drop in blood pressure with or without syncope, or an increase in the ventricular rate to above 130 beats per minute necessitate immediate withdrawal of the drug In the presence of severe myocardial damage, significant widening of the QRS complex must be considered as indication for stoppage of therapy

Quinidine is contraindicated during pregnancy We have seen a woman who aborted on several occasions when frequent bouts of tachycardia were treated with quinidine. In this case digitalis controlled the arrhythmia and a subsequent pregnancy was successfully culminated.

After the arrhythmia has been terminated, it is often necessary to continue maintenance doses of quinidine to prevent recurrences. Although the final maintenance schedule is determined by trial and error, in most instances the dosage required ranges between 0.2 and 0 4 Gm every four to six hours. Sokolow and Edgar[573] have found that a blood level of approximately 5 mgm per liter is required to prevent return of the arrhythmia Although further experience will be necessary to establish the usefulness of blood concentration estimation as a guide in quinidine therapy, it appears probable that this procedure will prove of value in determining maintenance doses necessary to prevent recurrences

Atabrine: Despite marked differences in chemical structure, atabrine closely parallels quinidine in respect to physiologic action on the heart[223, 615] In therapeutic doses atabrine is less toxic than quinidine, and several studies indicate that it is a promising agent in the therapy of cardiac arrhythmias.[392, 393, 608] The dosage recommended to terminate an arrhythmia is 0 1 Gm intravenously every two hours for five or six doses, 0.1 to 0 2 Gm doses are given orally to prevent recurrences. Gertler[224] found that atabrine, like quinidine, is most effective in arrhythmias not long established. Further observation will be required to determine the value of this new drug, especially since we know of one instance of death resulting from its intravenous use. It seems unlikely that atabrine will replace quinidine.

Magnesium Sulfate:[39, 453, 563, 594, 603] This drug acts both on the myocardium and on the conduction system. Since the magnesium ion is depressant to heart action,[241] beneficial effects should be expected in cardiac arrhythmias. Scherf[539] terminated three of eight attacks of paroxysmal tachycardia by using 10 to 15 cc. of magnesium sulfate in a 10 percent solution. With 20 cc. of the drug in a 20 per cent solution, all of 8 attacks of tachycardia were terminated; one instance of auricular fibrillation was unaffected. Zimdahl[645] terminated an auricular paroxysmal tachycardia with 22 cc. of a 25 per cent solution after lanatoside C, quinidine intramuscularly, Mecholyl, and 10 cc. of 25 per cent magnesium sulfate had failed.

The untoward effects of this therapy are sensations of intense heat, perspiration, nausea and general weakness Disturbed A-V conduction may occur and Scherf considers marked myocardial damage, intraventricular conduction disturbances, or gallop rhythm as probable contraindications Apparently the drug has a cumulative action so that repetition of the dose should be delayed for some time. Magnesium sulfate should not be used in renal failure. Magnesium ions are respiratory depressants as well as cardiac depressants.

Our experience with magnesium sulfate is limited, but we have not been impressed by the usefulness of the drug. Recent studies of other ions have stressed that sudden and drastic changes in ion chemistry are not without hazard. Winkler, Smith, and Hoff[660] have reported a fatality following injection of 30 cc. of a 25 per cent solution of magnesium sulfate. Enselberg[167] concluded that the therapeutic use of magnesium in arrhythmias was limited because of short action and occasional undesirable effects. However, this author had no experience with the drug in auricular tachycardia and had employed it in only two instances of flutter.

the ventricles are irritable as shown by the occurrence of multifocal premature ventricular systoles. In the past, conduction disturbances have been considered as contraindications to quinidine therapy. We believe that the drug may be used cautiously despite conduction disturbances.

The dosage of quinidine varies with different individuals and the exact amount required in a given instance can be determined only by trial and error. Sokolow and Edgar[573] and others[297, 298] have attempted to correlate the therapeutic effects with the blood levels of the drug. It has been demonstrated[391, 392, 393, 573] that the blood level of quinidine reaches a peak about two hours after oral administration. When the same dose is repeated at two-hour intervals the blood level gradually rises with each succeeding dose for the first four or five doses; however, the increment by which the blood concentration is increased grows smaller with each successive dose until finally, after the fifth dose, the blood concentration ceases to increase and the curve levels off unless the succeeding doses are substantially augmented. On the basis of these observations and our own clinical experience, the following *alternative* schedules are recommended:

(a) A dose of 0.4 Gm. is given initially and repeated every two hours until five doses have been given (total daily dose of 2.0 Gm.). If conversion to normal sinus rhythm is not obtained, this schedule is repeated on the following day, increasing the individual dose to 0.6 Gm. On the third day the individual dose is increased to 0.8 Gm (daily total of 4 0 Gm.). In our experience, the medical "custom" of giving 0.2 Gm. as a test dose is of no value and often entails unnecessary delay. Linenthal and Friedberg[392, 393] concluded that the evidence is insufficient to establish a causal relationship in the few reported instances of syncope, convulsions, collapse and respiratory difficulty following administration of single small doses of quinidine. Cinchonism and allergic reactions (rashes, purpura) are due to acquired sensitivities and develop after one to two weeks of quinidine therapy.

(b) Five doses are given at two-hour intervals as follows: 0.4 Gm., 0.6 Gm., 0.8 Gm., 1.0 Gm. and 1.2 Gm. If no serious toxic effects develop and the arrhythmia persists, this schedule is repeated daily for three successive days.

The parenteral use of quinidine is usually limited to emergencies, although in some instances of severe nausea or vomiting associated with an arrhythmia the physician will find the parenteral route of value. Several quinidine preparations are available for parenteral use; including quinidine hydrochloride, sulfate, lactate, and gluconate. Quinidine sulfate may be given intravenously by dissolving 0.2 Gm. of the powder in 20 cc. of distilled water, and administering slowly, or 4 Gm. may be dissolved in 500 cc. of saline or 5 per cent glucose and administered slowly by intravenous drip until the desired or toxic effect is obtained. Electrocardiograms should be taken during the drip and administration halted if toxic effects occur. The hydrochloride preparation is of value since it is commercially available in ampules of 0.5 Gm. in 5 cc. and is given as a 0.5 per cent solution. We have used primarily quinidine lactate which is marketed in ampules with 0.65 Gm. in 10 cc. of normal saline; this is diluted with 50 cc. of 5 per cent glucose for intravenous use and given slowly. The above preparations are of limited value for intramuscular use because of the pain involved in administering the quantity necessary to obtain adequate dosage. For intramuscular use the preparation quinidine hydrochloride 15 Gms., antipyrine 15 Gms , urea 20 Gms. and distilled water 100 cc. is especially valuable. A 2 cc. injection of this preparation will contain 0.3 Gms. of quinidine hydrochloride. Unfortunately, the preparation is unstable and cannot be stored for long periods.

In all instances, before a dose is given the patient should be examined to determine whether or not the arrhythmia has been terminated. If after three days the arrhythmia persists, it is unlikely that further quinidine therapy will prove successful.

ion, the various procedures available could be listed for each of the four auricular arrhythmias. Certainly the literature contains many excellent reviews on the therapy of the auricular arrhythmias[35, 36, 71, 187, 205, 344, 588, 607, 638] and many thorough analyses of specific aspects of the problem.[80, 287, 289, 317, 398, 460, 478, 481, 537, 576, 614, 624] But to be of maximum value to the practicing physician, a discussion of treatment should be applicable to the specific types of cases encountered clinically. In the present section we have attempted to provide such a guide to therapy. Certain clinical circumstances associated with the arrhythmias have been omitted because of their relative rarity; others undoubtedly have been overlooked. Nevertheless, the discussion covers the problems most commonly faced by the practicing physician.

AURICULAR PREMATURE BEATS

Auricular Premature Systoles Asymptomatic, with or without Heart Disease: Most premature systoles, whether auricular or ventricular, are asymptomatic. When such instances are discovered accidentally on physical examination or electrocardiograms, under no circumstances should the patient be informed of the irregularity nor should treatment be instituted. This is true whether or not the patient has heart disease. Each year many cardiac neurotics are created under such circumstances and patients take large amounts of quinidine needlessly.

Auricular premature systoles in patients with mitral stenosis frequently are believed to portend the onset of the more serious auricular arrhythmias. On the basis of such an assumption, prophylactic quinidine (0.2 to 0.4 Gm. every five hours during wakening) is often recommended. We believe that such a procedure is ill-advised in the vast majority of cases. In order to prevent properly this asymptomatic and benign disorder, it would be necessary to maintain an effective blood level 24 hours a day throughout the remainder of the patient's life. Nor should the physician interfere too much with the patient's joys of living by demanding the complete omission of tobacco, coffee, alcohol, etc. Also, the well meaning doctor may do more harm than good if he begins extensive and expensive investigation for a "trigger mechanism," such as diseased gallbladder. The treatment of any disorder which might be found, such as gallstones, would depend entirely upon the local condition and not on the presence or absence of extrasystoles.

Auricular Premature Systoles, Symptomatic, with or without Heart Disease: Palpitation is the usual symptom. The treatment can be considered 90 per cent psychotherapeutic and 10 per cent pharmacologic. Psychotherapeutically, various devices may be utilized by the physician. He can remind the patient that many doctors, perhaps he, himself, have premature systoles; and then, by mentioning symptoms that the patient has not yet described but probably has experienced, convince him that the doctor has a full understanding of the problem. His statement that the disturbance is not cause for concern then will be more readily accepted. The patient should be told that under no circumstances should he feel his pulse. Emphasize that the majority of patients with premature systoles have no symptoms, and tell him that his symptoms will gradually disappear. If the symptoms are mild, the patient should not be given quinidine on the first visit but should be assured that an effective drug exists and can be used if needed. After the physician has thoroughly explained to the patient that his disorder is functional and not a structural disease of the heart, good results will be obtained and most patients will not require further treatment.

In rare instances, usually in hypersensitive individuals, the presence of multifocal or frequent premature systoles necessitates pharmacologic treatment in addition to psychotherapy. Sedation in the form of phenobarbital in doses of 15 to 30 mgm. three or four times daily, or bromides in doses of 1.0 Gm. two or three times daily should be tried first. If these measures prove unsuccessful, quinidine sulfate (0.2 to 0.4 Gm.) four or five times daily may be admin-

Antihistaminics: McCawley and Dick[405] have recently used benadryl successfully in the treatment of auricular tachycardia, flutter and fibrillation. Experimentally, they and others[347, 348] have shown that the anti-histaminics have antifibrillatory properties in common with quinidine and procaine. Furthermore, benadryl possesses an advantage over quinidine in that in large doses it does not tend to produce ventricular arrhythmias or serious fall in blood pressure.[405, 418] This drug is administered in the following manner: A test dose of 50 mgm is given orally. Two hours later 200 mgm. diluted in 300 cc. saline solution is administered intravenously over a period of 30 minutes. If no results are obtained within six hours, 400 mgm. diluted in 500 cc. saline are given intravenously over a period of 45 minutes to an hour. If this treatment proves successful, maintenance doses are given orally.

Di Palma[143] has recently reviewed this problem in regard to other antihistaminics of similar structure which may be effective. Neoantergan was reported as being twice as active as quinidine against electrically induced fibrillation, while pyribenzamine was somewhat less active. Further clinical study with this group of promising drugs is certainly indicated, because of the combination of possible efficiency with low toxicity.

Fagarine: This drug has been claimed to yield good results in human auricular fibrillation[548, 599] Scherf[544] reported fagarine effective in stopping aconitine induced flutter and fibrillation. The drug has also been reported[133] to be five times more potent than quinidine in preventing electrically induced auricular fibrillation. Nevertheless, as Di Palma concluded,[143] the tendency of fagarine to produce ventricular fibrillation prevents its widespread clinical use, and any agent which replaces quinidine must be more potent but less toxic. Interest in fagarine nevertheless continues as a large group of synthetic derivatives based on the original structure proposed for this drug are being investigated[133] for antifibrillatory properties. The problem of finding the ideal antifibrillatory drug is exemplified by fagarine. Drugs may be found which are more potent than quinidine in raising the threshold of excitability but, as discussed in Chapter XVI, conductivity also may be more drastically depressed. Thus, undesirable cardiac effects which have been attributed to disturbed conductivity also probably will be magnified.

Miscellaneous: Auricular arrhythmias occurring during the course of various systemic afflictions are treated primarily by correction of the underlying disorder. Examples of such arrhythmias are those which occur during acute infections including pneumonia and childhood infectious diseases. Similarly, the arrhythmias which occur with digitalis intoxication are treated primarily by correction of the toxicity In the case of digitalis induced disturbances, magnesium[167] and potassium[168, 530] are reported to be unusually effective.

There are various procedures occasionally used in the therapy of auricular arrhythmias with which we have had no experience, and which at the present time are of unproven practical value. Toward such measures, the practicing physician should keep an open mind. These procedures include stellate ganglion blocks[103] and superior cervical sympathectomy. Procaine and its derivatives are said to be of limited benefit in the auricular arrhythmias while of great value in the ventricular disturbances. Dibenamine[242] appears to have little value in treating auricular arrhythmias but may prove of prophylactic value in patients, especially hyperthyroids, undergoing surgical procedures.

MANAGEMENT OF THE PATIENT WITH AN AURICULAR ARRHYTHMIA

As pointed out in the previous section, the treatment of auricular arrhythmias varies with the patient. If this were not true, a complete consideration of therapy need include only the general therapeutic procedures outlined above; the physician could be instructed to apply these procedures as he sees fit. Or, in cookbook fash-

ease or Presbycardia: In older patients, tachycardia may be fatal because it causes coronary insufficiency, shock states with peripheral stagnation, or pulmonary emboli. Unless there is response to the initial therapy, these patients should be hospitalized, if this can be accomplished without difficulty, so that they have the benefit of experienced nursing care and observation during their rapidly changing state. When the tachycardia is relatively slow, treatment is simpler as there is less shock and usually no failure From the beginning of the attack, the patient should be treated for congestive failure by digitalization whether or not this condition is present. If the rate is more rapid, treatment is more difficult as shock becomes more severe and the ectopic focus is more firmly established Shock, as evidenced by blood pressure and collapsed peripheral veins, must be treated by transfusions and sympathomimetic drugs such as paredrine. Congestive failure, as evidenced by full neck veins, must be treated by parenterally administered digitalis Carotid sinus stimulation should be used in these patients only with great caution because of the hazards associated with cerebral vascular disease. Ocular pressure may be tried. If the arrhythmia still persists, quinidine should be used Subsequently, Mecholyl should be tried and finally, parenteral quinidine with determination of blood levels The need for drastic therapy in these patients is confirmed by our experience, in the past three years, we have seen 3 deaths from tachycardia, all in the older age group.

Tachycardia in Infants: Here the auricular rate is extremely rapid, usually around 250 per minute and occasionally in excess of 300 Such arrhythmias bear a grave prognosis if allowed to continue untreated and undoubtedly account for many instances of death of infants for which no cause can be found. Difficulties in the diagnosis of tachycardia in infants are discussed in Chapter III. The internist, especially the cardiologist, must be familiar with this problem in order to aid the pediatrician.

The auricular paroxysmal tachycardia of infants is notoriously resistant to vagus-stimulating mechanical procedures and to various antiarrhythmic drugs including quinidine, Neostigmine, and Neosynephrine. The treatment of choice is digitalization which, fortunately, is almost always effective.[239] An initial dose of 2.5 cc. (0 5 mgm.) of Cedilanid or 0.05 Gm. of Digifolin is given intramuscularly. Subsequent dosage must be determined by the clinical response. The total dosage of digitalis required by different patients varies tremendously and will often be far in excess of that expected on the basis of the infant's weight. If the arrhythmia persists after digitalization, quinidine may be given simultaneously with maintenance doses of digitalis. Small doses of phenobarbital orally or of sodium phenobarbital intramuscularly may be administered for sedation. Gibson[229] reported successful results with Mecholyl in two infants who appeared moribund; 2.0 mgm were given intravenously without effect, but five minutes later conversion to normal sinus rhythm was achieved with a 14 mgm. dose.

Tachycardia Following Coronary Occlusion: In these instances the disorder usually is due to auricular infarction accompanying a posterior myocardial infarction[239] or to congestive failure accompanying the infarction.[9, 11, 402, 403 430] As pointed out by Askey[9] and others[432, 437] the combination of auricular tachycardia and myocardial infarction is rare.[312] The poor prognosis of patients with myocardial infarction in whom auricular tachycardia develops warrants heroic therapy. In such instances, the physician must stay with the patient day and night. The combination of shock and failure is common and the doctor must carefully evaluate all clinical signs, especially full neck veins in combination with empty peripheral veins and low blood pressure. If shock is present, treatment with transfusion and sympathomimetic drugs is indicated Digitalization and quinidinization must be used together for any heart failure. Frequently in these instances, time becomes of the essence, if

istered. Papaverine in doses of 0.065 to 0.2 Gm. three times daily and potassium acetate [530] 2.0 to 4.0 Gm. (30 to 60 grains) in a 25 per cent solution of peppermint water every four to six hours have been tried; in our experience these drugs have not proved too successful. The excessive use of alcohol, coffee and tobacco should be controlled, but strict omission of these items should be insisted upon only if benefit can be demonstrated. In most instances the institution of general measures such as weight loss or gain is indicated; proper exercise, fresh air, and relaxation will have more beneficial effect than specific medications.

AURICULAR PAROXYSMAL TACHYCARDIA

Functional Tachycardia, Asymptomatic, with Slow Rates: In these instances the cardiac rate is generally in the range of 90 to 100 The episodes last only for a few beats and the rates are usually so slow that the presence of the abnormality cannot be determined on physical examination and is accidentally discovered on electrocardiograms. The hearts are generally perfectly normal and the disturbance is frequently psychogenic or emotional in origin. We have seen two such cases in the past year. As in asymptomatic auricular premature systole, we believe it best not to inform the patient of the arrhythmia. The only treatment indicated, if any, is a sedative if the patient is aware of the disturbance. Strong reassurance as to the absence of heart disease is essential.

Functional Tachycardia, Symptomatic, with Fast Rates: This is the common type of auricular paroxysmal tachycardia. The cardiac rate is usually 180 or faster. The episodes may be brief and terminate spontaneously or they may last long enough to require drastic treatment. Frequent recurrence is common. The heart is usually normal, onset of the paroxysms may be psychogenic. Since these patients are usually extremely excited, the physician should first assure them that he knows exactly what the trouble is and that it is not serious. He should then try carotid sinus massage. It may be necessary to repeat the massage after sedation. If this procedure does not terminate the attack, and if the patient is in good general condition without shock and the heart is structurally sound, quinidine (0.4 Gm.) should be given together with a sedative sufficient to induce sleep. We usually recommend that the patient take pentobarbital (0.2 Gm.). In our experince, 95 per cent of the patients will awaken with the attack terminated. Whether the quinidine or the sedative causes the conversion is not known. If the tachycardia is still present when the patient awakens, we believe that Cedilanid is then the best therapeutic agent to use. In such cases, the drug should be given intramuscularly and followed by repeating the carotid sinus massage. If the condition still persists, it is wise to hospitalize the patient and ample quinidine should be given. Lastly Mecholyl should be used as described above. This procedure will result in successful conversion to normal sinus rhythm in 99 per cent of cases.

Rarely, adults exhibit paroxysms of tachycardia with extremely rapid rates, approximately 250 beats per minute. Carotid sinus massage and other vagal stimulants are usually ineffective in such instances. The treatment of choice in the absence of failure is quinidine, given parenterally (intramuscularly or intravenously) and with attention to blood levels. Failure or shock readily occurs in these patients and drastic therapy may be required If failure is present, intramuscular digitalis must be used simultaneously with the quinidine.

Tachycardia with Mitral Stenosis: Since these patients usually are aware of the presence of heart disease, sedation and reassurance that the disturbance will not be fatal is of prime importance. Cedilanid in combination with carotid sinus massage is the treatment of choice If ineffective, hospitalization probably is indicated and therapy will vary depending on the condition of the patient. If his condition is satisfactory, use quinidine and sedation, if critical, use Mecholyl.

Tachycardia with Arteriosclerotic Heart Dis-

ease or Presbycardia: In older patients, tachycardia may be fatal because it causes coronary insufficiency, shock states with peripheral stagnation, or pulmonary emboli. Unless there is response to the initial therapy, these patients should be hospitalized, if this can be accomplished without difficulty, so that they have the benefit of experienced nursing care and observation during their rapidly changing state. When the tachycardia is relatively slow, treatment is simpler as there is less shock and usually no failure. From the beginning of the attack, the patient should be treated for congestive failure by digitalization whether or not this condition is present. If the rate is more rapid, treatment is more difficult as shock becomes more severe and the ectopic focus is more firmly established Shock, as evidenced by blood pressure and collapsed peripheral veins, must be treated by transfusions and sympathomimetic drugs such as paredrine Congestive failure, as evidenced by full neck veins, must be treated by parenterally administered digitalis Carotid sinus stimulation should be used in these patients only with great caution because of the hazards associated with cerebral vascular disease Ocular pressure may be tried If the arrhythmia still persists, quinidine should be used Subsequently, Mecholyl should be tried and finally, parenteral quinidine with determination of blood levels The need for drastic therapy in these patients is confirmed by our experience, in the past three years, we have seen 3 deaths from tachycardia, all in the older age group

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the blood pressure is adequate digitalis and quinidine may be given intramuscularly, but if blood pressure is very low, the drugs should be given slowly intravenously. The diagnosis of auricular infarction is difficult and frequently impossible. However, if an electrocardiogram has been taken after the occlusion but before the onset of the tachycardia, careful perusal for Ta waves may give a clue as to the diagnosis.

AURICULAR FLUTTER

Flutter with Effective Block and Normal Ventricular Rate: In these instances the ventricles, *protected by auriculo-ventricular block, have a* near normal rate. Since the patient has no symptoms, no treatment is indicated. Such arrhythmias may continue for years without the appearance of symptoms.[102] The patient should be seen yearly unless symptoms occur.

Flutter with Inefficient Block and Rapid Ventricular Rate: Digitalis is the treatment of choice. The purpose here is to increase the auriculo-ventricular block and thus allow the ventricles to perform at efficient rates. Achievement of this result usually relieves symptoms. Sometimes digitalis will convert the flutter to fibrillation. It has been stated that upon withdrawal of the drug the fibrillation will convert to normal sinus rhythm. We have never observed this latter event, and Bloomfield's[45] review of this problem emphasizes the rarity with which it occurs. Occasionally digitalis will convert flutter, especially if of the paroxysmal type and with a relatively slow rate, to normal sinus rhythm.[572] If the flutter is slow, paroxysmal in origin, and conversion is desired, Mecholyl may be tried.[580] We have seen slow rate flutter in dogs converted to normal sinus rhythm with acetylcholine. Carotid sinus massage is usually ineffective in terminating the arrhythmia and tends to increase the auriculo-ventricular block.

After digitalis has slowed the ventricular rate and the patient is symptom-free, in almost all conditions it is advisable to continue on maintenance doses of digitalis Auricular flutter usually occurs in patients with arteriosclerotic heart disease and conversion to normal sinus rhythm is not indicated since relapse to flutter frequently occurs after a short period. However, if the heart is small and compensation good, conversion to normal sinus rhythm may be tried.

Auricular Flutter Associated with Organic Heart Disease: Auricular flutter with rheumatic heart disease is not common. If the myocardium is in good condition, the treatment should be conversion to normal sinus rhythm after digitalization. If the heart is large, the patient should merely be digitalized.

Auricular flutter, like tachycardia, may occur following a myocardial infarction. Most studies[9, 433, 457, 512] reveal that flutter is more frequent than tachycardia following myocardial infarction and bears a grave prognosis. Askey[9] found a 100 per cent mortality rate in such patients in whom the flutter persisted, and an 85.7 per cent mortality rate in those instances in which the ventricular rate was 120 or more. In these instances, time again is important; termination of the arrhythmia must be accomplished quickly. Optimum doses of digitalis and quinidine are recommended, intramuscularly or intravenously if the ventricular rate is rapid. Since these drugs are not without danger in the presence of myocardial infarction, no medication should be used if the ventricular rate is normal and no failure is present.

AURICULAR FIBRILLATION

Hyperthyroidism must be ruled out as an etiologic factor in each of the types of auricular fibrillation described below.

Functional Fibrillation with Slow Ventricular Rate: This is an infrequent arrhythmia, presenting no problem and requiring no treatment.[256] The patients come in for routine examinations without symptoms and without evidence of heart disease or previous thyroid disturbance. In such cases the physical examination is normal except for irregularity of the pulse confirmed by electrocardiogram to be slow fibrillation. We have observed three

patients who have exhibited this arrhythmia for 20 years or more without incapacity. As noted in Chapter XII, cinematographic studies show that the fibrillating auricle empties fairly well with little stagnation and with a substantial ventricular inflow tract. When such an auricle was digitalized, however, decreased motion could be seen The fact that digitalis causes stagnation of blood, together with the suggested effect on clotting time,[141, 198, 202, 438, 472] may increase the possibilities of thrombi Thus, nothing is gained by digitalization in this asymptomatic type of fibrillation and the drug conceivably could do some harm. By advising against conversion, we do not imply that fibrillation is as satisfactory as normal sinus rhythm (Chapter I). Despite the fact that presumably cardiac output is decreased in fibrillation, if the patient has no symptoms, a slow heart rate and a small heart, he need not be treated. The physician should not make a cardiac neurotic of the patient. Provided he remains asymptomatic, yearly examinations should suffice.

Functional Fibrillation with Fast Ventricular Rate: This disorder requires drastic treatment, as failure may occur quickly. Rapid digitalization to slow the ventricular rate may be lifesaving Conversion to normal sinus rhythm can then be accomplished We have seen conversion in the presence of profound failure, the patient has now been well for 10 years[66, 69] Phillips and Levine[186] considered a group of patients without evidence of heart disease but in failure as a result of fibrillation. These workers recommended conversion to normal sinus rhythm early in the course of the arrhythmia before an irreversible stage of failure has been reached.

Fibrillation with Arteriosclerotic Hypertensive Heart Disease or Presbycardia: This is the most common cause of auricular fibrillation It is usually found in elderly patients, and although overt failure may not be present there is always some degree of incipient failure The treatment consists of digitalization, it is well to remember that in senile patients fractional amounts of the usual doses of digitalis may be of great help. Conversion to normal sinus rhythm should not be attempted since recurrence of the fibrillation after a short time is probable.

In rare instances in the absence of hyperthyroidism, all attempts to slow the ventricular rate prove futile and the patient remains in failure. In such instances, it is best to convert the fibrillation to normal sinus rhythm after which compensation may take place and the arrhythmia will not recur.

Auricular fibrillation is the most frequent auricular arrhythmia in patients with acute myocardial infarction.[7, 9, 419, 457, 512] Askey[7] has reviewed the therapy of this problem; our experience is in agreement with the therapeutic regimen recommended. When fibrillation appears with the acute episode, conversion should be done immediately. If the fibrillation appears several days after the infarction, Askey advises a delay of 12 hours to determine if the patient will convert spontaneously. If failure is present, treatment with quinidine and digitalis is indicated

Fibrillation with Mitral Stenosis without Failure: Digitalization is the preferred therapy in most instances since the arrhythmia often recurs following conversion to normal sinus rhythm. Nevertheless, competent cardiologists of our acquaintance uniformly try to restore normal sinus rhythm and attempt to prevent recurrence with maintenance doses of quinidine. Our only objection to this procedure is that if fibrillation recurs, as we find it often does, either unavoidably or due to failure of the patient to take the quinidine, the arrhythmia returns at an extremely rapid and disastrous rate.

Conversion is indicated under two circumstances. (1) if the patient is normally well compensated, the heart is comparatively small and failure comes on with the presence of fibrillation, and (2) if by history the attack is paroxysmal In such instances conversion should not be sought so diligently that the patient is endangered For example, if conversion has not occurred after large doses of quinidine, the drug should be discontinued and the patient

digitalized. Also, if the rhythm converts back and forth several times, it is best to digitalize the patient and discontinue attempts to obtain *permanent conversion*.

Fibrillation with Mitral Stenosis with Failure: *These patients have enlarged hearts with* large auricles. Digitalization *is the treatment* of choice and may frequently be life-saving. In this disorder the ventricular rate will serve as a better guide to effective digitalization than in normal sinus rhythm. Conversion to normal sinus rhythm with quinidine usually should not *be attempted*.

Fibrillation with Organic Disease with Embolization (Usually Mitral Stenosis): We agree with Sokolow[571] that in a few selected cases the treatment is to convert the fibrillation to normal sinus rhythm if possible, despite the danger than an embolus will develop at the time of conversion.[7] In many instances the embolization stops after conversion has taken place. If *attempts to obtain conversion are unsuccessful*, treatment with anticoagulants (Dicumerol) is indicated.[112, 200, 280A, 577, 678] If the patient continues to have emboli from the appendix, atrial appendectomy[424] is a necessity since an embolus to the brain will probably maim or kill the patient. In the future the treatment of choice would appear to be an appendectomy in patients whose thrombi form in the auricular appendix. *Less danger exists when the thrombi* form in the body of the left auricle since this area is less contractile and less active than the appendix (Chapter I).

Surgical Therapy in Mitral Stenosis: In recent years surgical therapy *for mitral stenosis* has been gaining importance. Certainly *this may be the ultimate answer to the problem* since no curative medical treatment is available. Atrial appendectomy to prevent embolization has already proved of value in a few cases. Commissurotomy is promising as the definitive therapy for mitral *stenosis. At the present time* we do not consider the presence of fibrillation *alone to be an indication* for commissurotomy, since such a complication may be well handled medically. But if appendectomy is to be performed, the advisability of commissurotomy should be considered since this operation cannot be done at a later date. The indications for commissurotomy have not been completely defined but are discussed by Bailey[17] and Harken.[260] *With greater use and improved techniques, the operation undoubtedly will become* a more popular form of therapy.

Since patients with mitral stenosis are initially under the care of an internist, the training of the surgeon selected to perform these procedures should be considered. It is erroneous to believe that even an excellent chest surgeon can perform intracardiac surgical operations without *special training. The finest of surgeons* working under excellent conditions have had mortality rates well over 50 per cent in their early cases, while later they have reduced the rate to 10 per cent. Furthermore, many patients who survived the operation may not have been benefited or may have been harmed. Thus it is *apparent that special training is invaluable.* Because of the limited number of subjects on whom the operations can be performed, relatively few chest surgeons need be able to do these procedures. Those interested enough to undergo special training should obtain it from the men who devised these procedures and have *worked on them for years. The novice in cardiac* surgery, no matter how able in other types of surgery, should profit from the experience of the master when mortality can be so greatly reduced by training. It is to be remembered that slight errors during a herniorrhaphy may be of little or no importance while in cardiac surgery *such errors often are disastrous*

Fibrillation with Thyrotoxicosis: The fundamental treatment here consists of attacking the hyperthyroidism. We recommend radioactive iodine for routine cases If failure is present it usually indicates the existence of an underlying heart disease (Chapter XI) which must also be *treated. In the interval of weeks or months* before the radioactive iodine becomes maximally effective, digitalization may be necessary The physician should remember that during this interval the rapid ventricular rate cannot be

slowed to normal, rates around 90 are satisfactory. If surgical therapy is used, the preoperative preparation (propylthiouracil, potassium iodide, sedatives, etc.) should include digitalization. Conversion to normal sinus rhythm is accomplished by quinidine after the establishment of a near normal basal state by either surgical or anti-thyroid medical therapy.

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concept of the unitary nature of the auricular arrhythmias is then outlined and the supporting observations are summarized.

The four auricular arrhythmias produced by stimulation of a single site on the auricle exhibit the following cinematographic, electrocardiographic and oscillographic features:

Auricular Premature Systoles: When the rate at which stimuli are discharged from an experimentally produced ectopic focus is less than the rate of discharge from the sinus node, isolated premature systoles occur. The contraction wave initiates at the ectopic focus and spreads throughout the contractile portions of the auricles in all directions simultaneously. The premature beat is identical in all respects to that of normal sinus rhythm except for its ectopic site of origin and probably a slightly slower rate of propagation. The configuration of the auricular deflection is determined by the site of origin of the beat; it may be identical with the auricular complex of sinus rhythm if the ectopic focus is at or near the sinus node

Auricular Paroxysmal Tachycardia: When the rate of discharge from an ectopic focus exceeds the rate of discharge from the sinus node, the ectopic focus becomes the cardiac pacemaker and auricular paroxysmal tachycardia results The appearance of the contraction wave is identical with that of premature systoles; it takes origin in the ectopic focus and spreads throughout the contractile auricular musculature in all available directions simultaneously. The ventricles usually respond to each auricular beat Cinematographically the increase in auricular rate is associated with a decrease in the speed of propagation of the contraction wave over the auricles. The auricular deflection is identical with that of premature systoles from the same focus

Auricular Flutter: When the rate of discharge of stimuli from the ectopic focus falls in the range of approximately 300 to 400 per minute,* auriculo-ventricular block generally occurs and auricular flutter is said to be present. The site of origin and course of the contraction wave is similar to that of auricular paroxysmal tachycardia and of premature systoles; each wave arises at the ectopic focus and spreads in all available directions simultaneously. Again the inverse relationship between the auricular rate and the speed of propagation of the contraction wave prevails, resulting in a further shortening of the diastolic period. The long duration of systole and the extremely short diastole in flutter are responsible for the undulating, restless electrocardiographic appearance characteristic of the arrhythmia. This undulatory pattern is believed to consist of a P' deflection (wave of depolarization) followed by a bowed Ta wave (wave of repolarization) (Chapter VII). A similar pattern is recorded during rapid auricular tachycardia with auriculo-ventricular block. Thus, it is apparent that "auricular flutter" is simply a variant of tachycardia in which the rate has become so rapid that physiologic block has appeared, and in which the focus is usually in the caudal end of the auricles.

Auricular Fibrillation: Auricular fibrillation ensues when the rate of discharge from an ectopic focus exceeds the rate at which the auricles are able to respond rhythmically. This "fibrillation threshold" is usually between 400 to 600 per minute.* Like the previously described arrhythmias, fibrillation is initiated in a single ectopic focus The motion in the fibrillating auricle, however, is entirely different from that observed in the slower-rate rhythms. Microscopic ("M") activity occurs constantly throughout the contractile auricular musculature; this phenomenon is seen in no other auricular arrhythmia. Against the background of chaotic activity, macroscopic ("L") waves are distinguished which differ from the contraction waves of the previously described arrhythmias in two essential respects: (a) they do not necessarily arise from the initiating focus but may spring from any part of the auricle, and (b) they travel in diverse directions and frequently change course from one direction to

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CHAPTER XVIII

The Unitary Nature of the Auricular Arrhythmias

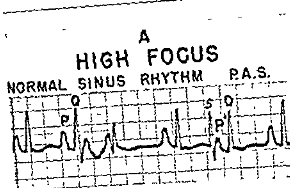
THE SCIENTIST *constantly strives to explain a* diversity of observations in terms of as few fundamental principles as are consistent with the known facts. To this end, the study reported in this monograph has been designed to explore not only the character of each of the four auricular arrhythmias but also their basic similarities and differences. The present chapter is devoted to a marshaling of observations, both experimental and clinical, concerning the relationship between auricular premature systoles, tachycardia, flutter and fibrillation. From these observations has evolved the concept of the unitary nature of the auricular arrhythmias.

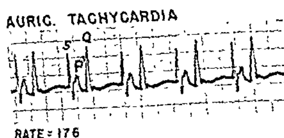
That a close relationship exists between the various auricular arrhythmias has long been suspected clinically and was proposed by certain members of the Viennese school of cardiology at the turn of the century. In 1912 Lewis,[359, 361] as a result of extensive clinical investigation, advanced the following theory of the kinship of the arrhythmias: "In an article written in conjunction with Dr. Schleiter, I have already drawn attention to the close relation between paroxysmal tachycardia of auricular origin and auricular fibrillation. The conclusion that they are part and parcel of the same pathological process was supported by a number of facts, none of which were more noteworthy than the frequent occurrence of the two conditions in the same patient, and especially the immediate passage of one to the other. The argument applies even more strongly to auricular flutter, for in 13 cases of the present series auricular fibrillation occurred in seven, and in six, one condition passed directly into the other. The separate mechanisms must be very closely related; and it is probable that they have a similar pathogeny. The occasional occurrence of tachycardia of a more simple form (Case 2) and the frequent occurrence of single premature beats in the same patients when a sinus rhythm has been re-established (they were present in five out of the six cases in which the normal mechanism was observed) seems to connect flutter with the remaining auricular mechanism in which disturbing beats are found The conclusions that

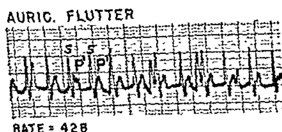
1. Single premature auricular contractions.
2. Small groups of the same.
3 Paroxysms of tachycardia from single auricular foci.
4. Auricular flutter.
5 Paroxysms of tachycardia from two or more auricular foci.
6. Auricular fibrillation.

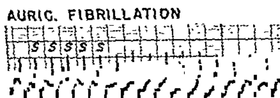
arise essentially in the same manner, namely, through the pathological or heterogenetic origin of new impulses in the auricle, are clearly suggested by the facts at our disposal." Subsequently, Lewis[383] abandoned this view and attributed to auricular flutter and fibrillation a mechanism different from that common to auricular premature systole and tachycardia.

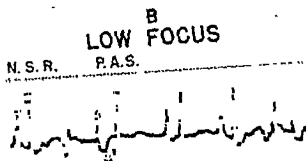
Despite widespread awareness among clinicians of the close relationship between these disorders, convincing evidence of their common origin has not hitherto been advanced. We believe our experimental study provides such evidence. For purposes of review, the essential features of each of the experimentally produced auricular arrhythmias is briefly restated. The

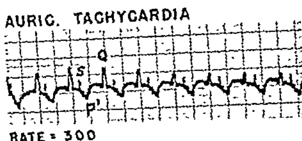


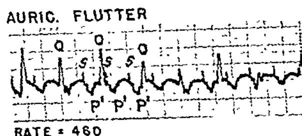












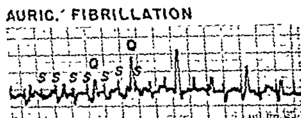


Figure 308 Production of four auricular arrhythmias by electrical stimulation
(A) Foc[illegible]
(B) [illegible]
Whe[illegible]
rhythm o[illegible] premature systoles (PAS) occur Tachycardia ensues when rate of stimulation exceeds rate of sinus rhythm. Flutter occurs with progressive increase in rate and development of auriculo-ventricular block At extremely rapid rates of stimulation auricular fibrillation is present. Note that P' wave of arrhythmias from focus high in the auricle is always upright, conversely P' wave in arrhythmias from low focus is always inverted Electrocardiograms taken at double speed.

stimulation is increased to a range of 150 to 350 per minute, the cinematographic characteristics of auricular paroxysmal tachycardia appear At rates of stimulation between approximately 350 and 400 per minute, the films represent auricular flutter Rates above 400 stimuli per minute evoke auricular fibrillation.

Electrocardiograms taken during such an experiment demonstrate that progressively increasing rates of electrical stimulation at a single site on the auricle of a given animal may evoke all four auricular arrhythmias seriatim (Figure 308). Thus the mode of origin of the auricular arrhythmias as determined cinemato-

another. The electrocardiographic and oscillographic appearance of fibrillation also differs markedly from that of other auricular arrhythmias. This difference is particularly evident on oscillographic tracings in which small deflections occurring at rates up to 20,000 per minute are inscribed. Such deflections are not present in the other rhythms and probably represent the electrical counterpart of the microscopic activity seen in the motion picture.

The auricles are capable of orderly contraction through a wide range of rates. Unlike the ventricles, they have no organized conducting system. In normal sinus rhythm, the impulse starts in a single cell or group of cells in the sino-auricular node and is propagated from cell to cell, spreading in an orderly wave-like manner in all directions. In auricular premature systole, auricular paroxysmal tachycardia and auricular flutter, the impulses are formed in an ectopic focus from which the contraction waves spread in all directions, again traveling from cell to cell in orderly sequence. When the rate of discharge from the ectopic focus reaches the fibrillation threshold, conduction fails and the rule of orderly transmission from cell to cell is broken. Auricular fibrillation is then present.

The close relationship between the experimentally produced auricular arrhythmias is implicit in the above description of these disorders. The four arrhythmias have a common mode of origin, namely, the emission of stimuli from an ectopic focus in the auricles

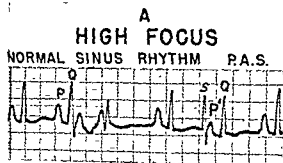
In the experimental animal, when all other circumstances are controlled, which of the four arrhythmias will prevail is determined solely by the rate of discharge from the ectopic focus. If the rate is less than that from the sinus node, auricular premature systoles will occur. If the rate exceeds that from the node, the ectopic focus becomes the cardiac pacemaker In a given experimental animal, if the normal sinus rate is 100 per minute, when the ectopic focus discharges at a rate of approximately 150 to 250 per minute, auricular paroxysmal tachycardia results. At rates of 250 to 400 per minute, auricular flutter is usually present. When the rate exceeds 400 to 600 per minute, the auricles usually are no longer able to respond rhythmically and auricular fibrillation results. Clinically and experimentally, this sequence is modified by a number of factors including age, vagal tone, anoxia, location of the ectopic focus, conductivity, and the general health and nutritive state of the auricular musculature.

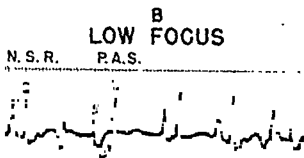
The contraction waves and electrical impulses in auricular premature systole, auricular paroxysmal tachycardia and auricular flutter initiated at the same ectopic focus are identical in site of origin and course of propagation; they consistently arise at the focus and spread outward in all available directions through the auricular musculature. The distinguishing cinematographic and electrocardiographic features of each of these three arrhythmias develop gradually as the rate of discharge from the focus progressively increases or decreases, indicating that no basic change in mechanism occurs during the transition. Only when the fibrillation threshold is reached (at a rate of discharge of approximately 500 impulses per second) do unique characteristics appear abruptly, in the form of chaotic mechanical and electrical activity occurring in minute muscle segments throughout the auricles. Whatever the precise nature of the electro-biochemical changes underlying these phenomena, the chaotic activity in the fibrillating auricle is directly related to the rapid rate of discharge of stimuli from the ectopic focus.

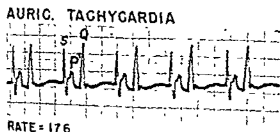
This is the concept of the unitary nature of the auricular arrhythmias. It is derived from the following 11 observations.

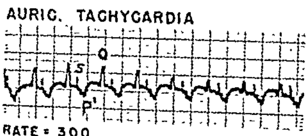
Observation 1. Effect on Auricular Rhythm of Progressive Increases in the Rate of Electrical Stimulation of the Auricles

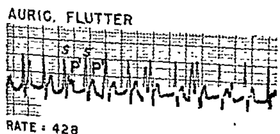
Single induction shocks at progressively increasing rates are sent into the auricles at a single focus while high speed cinematographs of the auricles are recorded. When the rate of electrical stimulation is less than the rate of discharge from the sinus node, the films reveal occasional premature systoles. As the rate of

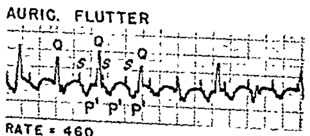


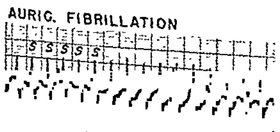












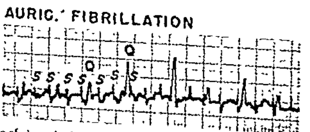


Figure 308 Production of four auricular arrhythmias by electrical stimulation
(A) Focus is in cephalic region of auricle
(B) Focus is near inferior vena cava
When rate of stimulation (S) is less than rate of sinus rhythm only scattered auricular premature systoles (PAS) occur Tachycardia ensues when rate of stimulation exceeds rate of sinus rhythm. Flutter occurs with progressive increase in rate and development of auriculo-ventricular block At extremely rapid rates of stimulation auricular fibrillation is present Note that P' wave of arrhythmias from focus high in the auricle is always upright, conversely P' wave in arrhythmias from low focus is always inverted Electrocardiograms taken at double speed.

stimulation is increased to a range of 150 to 350 per minute, the cinematographic characteristics of auricular paroxysmal tachycardia appear. At rates of stimulation between approximately 350 and 400 per minute, the films represent auricular flutter. Rates above 400 stimuli per minute evoke auricular fibrillation

Electrocardiograms taken during such an experiment demonstrate that progressively increasing rates of electrical stimulation at a single site on the auricle of a given animal may evoke all four auricular arrhythmias seriatim (Figure 308). Thus the mode of origin of the auricular arrhythmias as determined cinemato-

graphically and electrocardiographically is identical; the rate of stimulation determines which arrhythmia is produced.

Observation 2: Relation of Aconitine Concentration to the Production of the Auricular Arrhythmias

On repeated occasions we have produced all the auricular arrhythmias in a single animal by applying various concentrations and quantities of aconitine to a small circumscribed area on the dried surface of the auricle. Considerable variation in sensitivity exists from animal to animal. Our impression is that in many instances 0.05 per cent aconitine applied in this manner results in the production of scattered auricular premature systoles, 1 per cent aconitine produces auricular paroxysmal tachycardia, a 1.5 to 2.0 per cent solution evokes auricular flutter, and 3 to 10 per cent aconitine initiates auricular fibrillation. The production of auricular fibrillation in young animals has in our experience sometimes been difficult. Any of the arrhythmias may be produced by repeated application of weaker aconitine solutions.

The cinematographic appearance of an auricular arrhythmia from a given focus discharging at a given rate is identical whether the disorder is initiated by aconitine application or electrical stimulation. That is, each of the four auricular arrhythmias produced by varying the concentration of aconitine applied to the focus is cinematographically identical with its counterpart in the series of arrhythmias produced by varying the rate of electrical stimulation. Thus the conclusion seems reasonable that a given concentration of aconitine has the same ultimate effect on the auricular musculature as a given rate of electrical stimulation. Presumably a low concentration of aconitine, like a low rate of stimulation, occasions the formation and discharge of relatively few impulses at the ectopic focus; the discharge of these impulses results in premature systoles. Higher concentrations of aconitine, like more rapid rates of electrical stimulation, set up a rapidly discharging focus and the more rapid-rate arrhythmias are obtained. That mere variation in the concentration of the exciting agent produces four basically different disorders would appear unlikely. A more reasonable interpretation of the above observations is that the four auricular arrhythmias are forms of the same fundamental disturbance.

Observation 3: Effect of Temperature Changes on the Aconitine Focus

Aconitine is applied to the surface of the auricle in a concentration sufficient to produce auricular fibrillation. Continuous direct auricular and indirect limb lead electrocardiograms are taken. When fibrillation is established, the aconitine focus is cooled by ethyl chloride spray or by the direct application of ice. As the cooling progresses, a transition from fibrillation to flutter usually is recorded, followed by the successive appearance of auricular paroxysmal tachycardia, premature systoles and finally sinus rhythm. Frequently, one or all of the rhythms intermediate between fibrillation and sinus rhythm fails to appear.

When the cooling is discontinued and the aconitine focus is allowed to thaw, the arrhythmias usually recur in the reverse order. Not infrequently, one or more of the rhythms intermediate between sinus rhythm and fibrillation may occur too briefly to be distinguished or may be absent. Occasionally, an arrhythmia other than fibrillation persists for a prolonged period of time. By the judicious application of aconitine and cooling agents, any desired arrhythmia usually can be produced and maintained for periods long enough to permit adequate study.

As is true of chemical phenomena in general, the biochemical and electrical activity initiated by the aconitine probably is decreased by the cooling process, which in turn diminishes the rate at which stimuli are emitted from the focus. Conversely, warming increases the chemical activity of the focus, thus increasing the rate of discharge of stimuli. In terms of the ultimate effect on auricular function, varying the temperature of the focus is comparable to varying

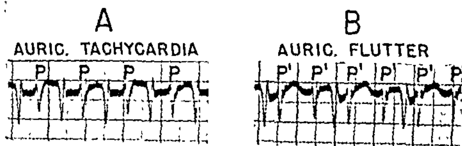


Figure 309 Direct auricular lead electrocardiogram recorded as rate of stimulation at single ectopic focus (aconitine) is increased to produce transition from tachycardia (A) to flutter (B) Note that P' waves of the two arrhythmias are almost identical.

the concentration of aconitine or the rate of electrical stimulation Each method alters the rhythm by increasing or decreasing the rate of discharge from the focus. A similar effect occurs when the temperature on the sinus node is altered; cooling the node slows the cardiac rate while the application of heat to the node results in marked cardiac acceleration

Observation 4 Cinematographic Appearance of the Experimentally Produced Arrhythmias

In high speed cinematographs of the auricular arrhythmias, the contraction waves of premature systoles, auricular paroxysmal tachycardia and flutter are essentially *identical in* appearance. Each starts at the ectopic focus and travels through the auricles in all directions simultaneously When a *single* auricular contraction is viewed, the observer cannot determine whether the prevailing arrhythmia is auricular premature systole, auricular paroxysmal tachycardia or flutter. Only the rates at which the contraction waves arise and traverse the auricle *differentiate* the cinematographic appearance of the three arrhythmias. The *motion* in the fibrillating auricle differs from that seen in the slower rate rhythms

Observation 5. Configuration of the Auricular Deflections in Direct Auricular Lead Electrocardiograms of the Experimentally Produced Arrhythmias

In innumerable instances direct auricular lead electrocardiograms were recorded while the four auricular arrhythmias were produced from a single ectopic focus. These tracings consistently show that in a given animal the configuration of the P' waves recorded in direct auricular leads is identical in premature systoles, auricular paroxysmal tachycardia and flutter (Figure 309). In each of these arrhythmias a pure negative deflection is recorded from electrodes at the focus; the positive portion of the deflection becomes larger as the distance between the electrode and the focus is increased. Thus the site of origin and course of the electrical impulse is identical in these three arrhythmias; it arises at the focus and spreads outward through the auricular musculature. This finding parallels our cinematographic observation concerning the identical site of origin and course of the contraction waves of premature systoles, tachycardia and flutter from the same focus. As might be anticipated from the cinematographs, the onset of fibrillation is marked by a distinct change in the configuration of the auricular complexes in direct lead electrocardiograms.

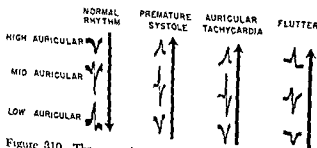


Figure 310 Three simultaneous esophageal leads from behind various parts of auricle during normal sinus rhythm, auricular premature systole, auricular tachycardia and auricular flutter. The intrinsicoid deflections are identical in all tracings during the three arrhythmias, the ectopic focus is low in the auricles and the impulse travels upward. During normal sinus rhythm the impulse starts at sino-auricular node and travels downward

Observation 6: Configuration of the Auricular Deflections in Esophageal Lead Electrocardiograms of the Clinical Auricular Arrhythmias

The shape of the auricular deflections of auricular premature systole, auricular tachycardia and flutter in esophageal lead electrocardiograms (Figure 310) recorded from man resemble those in direct lead electrocardiograms from experimental animals. Records from electrodes at the focus exhibit a pure negative deflection; the deflection becomes more positive as the electrode is moved away from the focus. This observation suggests that the site of origin and course of the electrical impulse in these three arrhythmias from the same focus is similar whether the disturbance is spontaneous or experimentally produced

Observation 7: Configuration of the Auricular Deflections in Indirect Lead Electrocardiograms of the Experimentally Produced and Clinical Auricular Arrhythmias

Auricular premature systoles are produced by the local application of aconitine to the wall of the auricle. Continuous electrocardiograms recording limb leads 1, 2 and 3 are taken as the rhythm spontaneously changes from auricular premature systoles through auricular paroxysmal tachycardia and auricular flutter to fibrillation (Figure 311).

As the auricular rate progressively increases, the configuration of the auricular complexes undergoes a progressive change from the narrow, sharp deflections of auricular premature systoles to the characteristic undulatory pattern of flutter. During the transition from auricular premature systole to auricular paroxysmal tachycardia the auricular deflection retains its same general contour (Figure 311A and B). As the auricular rate continues to increase a Ta wave appears and gradually grows larger until finally the typical flutter undulation appears (Figure 311C and D). The isoelectric interval between auricular deflections becomes progressively shorter and finally may disappear with the development of the undulatory pattern. No abrupt change in the contour of the auricular deflections occurs during the transition from auricular premature systole through auricular tachycardia to flutter, indicating that the electrocardiographic differences between these arrhythmias reflect differences in degree rather than in mechanism of the fundamental disturbance.

Limb lead and chest lead electrocardiograms of the clinical arrhythmias exhibit a similar gradual transition in the configuration of the auricular deflections. The P′ waves are similar in tracings of auricular premature systoles, slow auricular paroxysmal tachycardia, fast auricular paroxysmal tachycardia and flutter. The main differences consist of the occurrence of Ta waves at rapid auricular rates and the development of auriculo-ventricular block in flutter.

We have studied many tracings from patients in which two or more arrhythmias are represented. Only when fibrillation develops does the shape of the P′ wave change.

Observation 8. The Action of Drugs on the Experimentally Produced and Clinical Auricular Arrhythmias

The mode of action of quinidine and digitalis on the auricular arrhythmias provides collateral evidence of the unitary nature of these disturbances Clinically, quinidine may terminate any of the auricular arrhythmias and restore normal sinus rhythm. Experimentally, the intravenous administration of quinidine often converts fibrillation to sinus rhythm via a gradual transition through flutter, auricular paroxysmal tachycardia and auricular premature systoles (Figure 297, Chapter XVI).

A similar transition has been obtained with digitalis, although this drug more frequently fails to evoke one or more of the intervening arrhythmias. Sokolow and Chamberlain [572] have made similar observations in man.

Observation 9: Incidence of the Clinical Arrhythmias

The concept that the auricular arrhythmias represent different degrees of severity of the

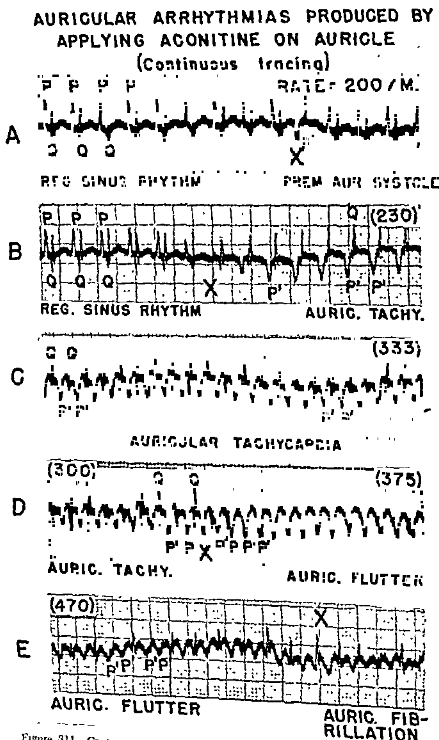


Figure 311 Continuous tracings, lead 2, of all four auricular arrhythmias produced by one application of aconitine at caudal region of right auricle

(A) Premature systole is present with inverted P' wave marked X

(B) Onset of auricular tachycardia at rate 230

(C) Tachycardia rate reaches 333 Small Ta waves appear

(D) At rate 375 flutter occurs

(E) Flutter is well established at rate 470 (voltage on electrocardiograph lowered) At X fibrillation threshold is passed and auricular *fibrillation* appears. Thus all four auricular arrhythmias may be produced from the same focus

Observation 6: Configuration of the Auricular Deflections in Esophageal Lead Electrocardiograms of the Clinical Auricular Arrhythmias

The shape of the auricular deflections of auricular premature systole, auricular tachycardia and flutter in esophageal lead electrocardiograms (Figure 310) recorded from man resemble those in direct lead electrocardiograms from experimental animals. Records from electrodes at the focus exhibit a pure negative deflection; the deflection becomes more positive as the electrode is moved away from the focus. This observation suggests that the *site of origin and* course of the electrical impulse in these three arrhythmias from the same focus is similar whether the disturbance is spontaneous or experimentally produced.

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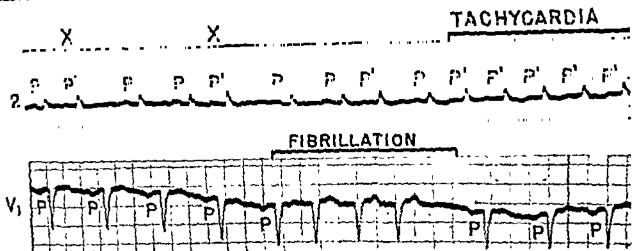
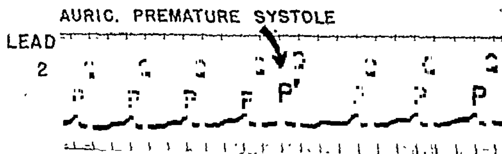
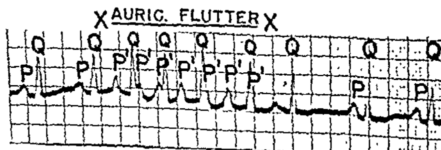


Figure 313. Continuous electrocardiograms from patient showing auricular premature systole and auricular tachycardia in lead 2 and auricular fibrillation in lead V₁. Note P' wave is same in premature systole as in tachycardia.





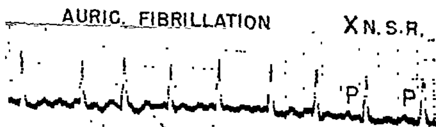


Figure 314 Continuous cardiograms showing premature auricular systoles, short bouts of auricular flutter and fibrillation. Note P' wave is the same in auricular premature systoles and auricular flutter.

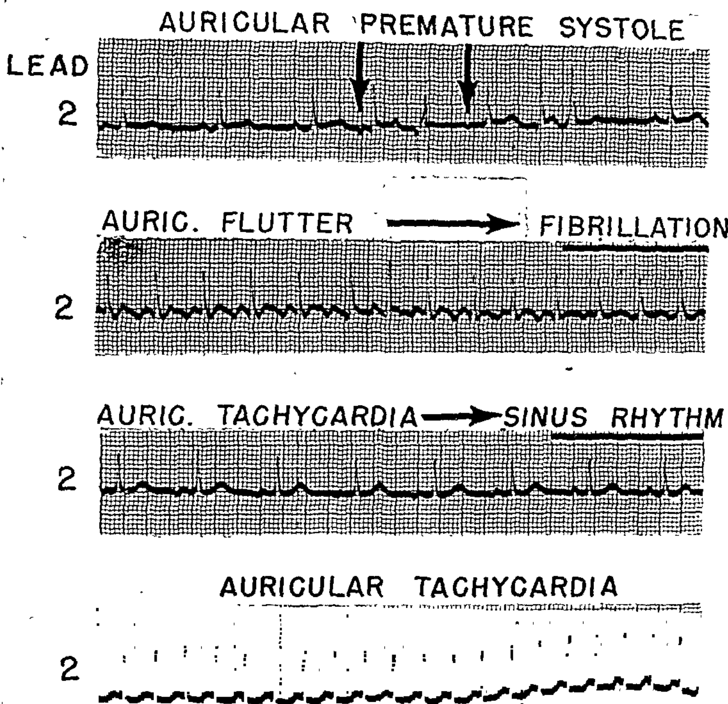
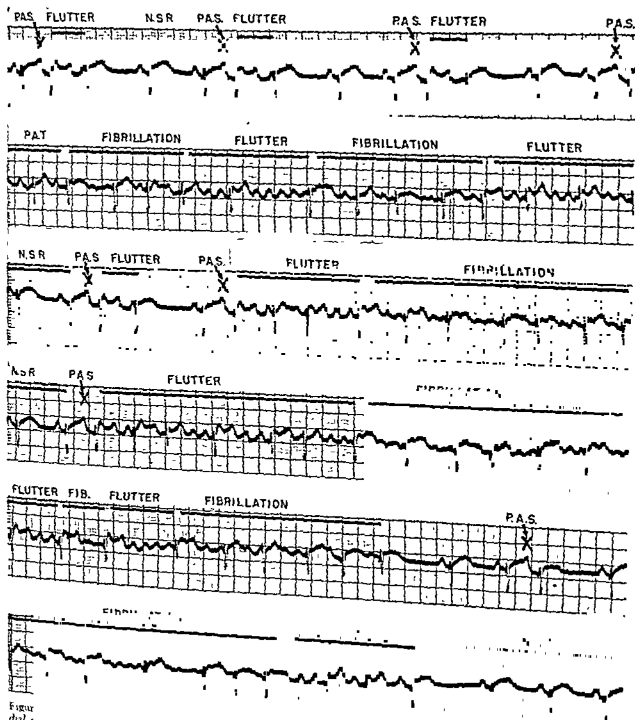


Figure 312 Auricular arrhythmias following catheterization of heart Upper 3 tracings from a single patient exhibiting premature auricular systoles, auricular tachycardia, flutter and fibrillation Lower tracing taken from another patient during cardiac catheterization demonstrates auricular tachycardia (Courtesy of Dr D M Green)

same fundamental disturbance is consistent with the known facts concerning the relative clinical frequency of these disorders. Thus auricular premature systole, presumably the mildest form of the disturbance, is the most common of the arrhythmias and occurs with greatest frequency among young and healthy individuals. On the other hand, in diseased hearts and in aged persons, where the specific disturbance is apt to be more severe and the fibrillation threshold lower, the incidence of auricular fibrillation is greatest. Auricular paroxysmal tachycardia occupies an intermediate position with regard to the severity of the fundamental disturbance, hence it is less common than auricular premature systoles among young and healthy individuals, and less common than auricular fibrillation among aged persons and in diseased hearts. When the disturbance is more severe than in tachycardia, a circumstance most likely



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auricl

...cephalic region of the auricle became irritable and acted as ectopic pacemaker. Within a period

the tracings. It is unlikely that four disorders of fundamentally different mechanisms could occur in a period of three minutes from an irritable auricular muscle.

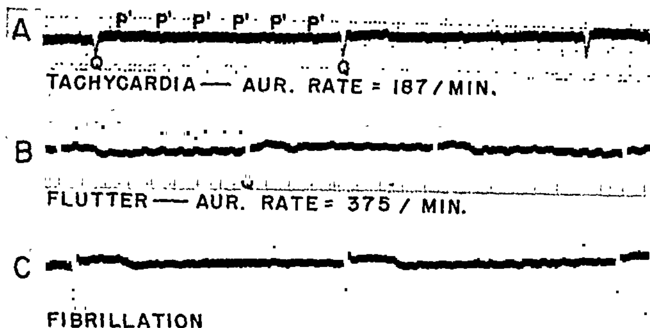


Figure 315 Electrocardiograms from patient with complete heart block. The presence of the block facilitates identification of deflections

(A) At auricular rate 187 per minute the deflections are characteristic of auricular tachycardia

(B) Rate increased to 375 per minute The tracing resembles flutter.

(C) Fibrillation is present

to occur in association with advanced age or disease, auricular flutter results. However, flutter is the rarest of the auricular arrhythmias because it occurs in a very narrow rate range and because it frequently converts to fibrillation due to lowering of the fibrillation threshold in aged persons and in diseased hearts. Flutter is usually the most difficult arrhythmia to produce and maintain during animal experiments.

Observation 10: Production of Arrhythmias by Mechanical Stimulation of the Heart in Experimental Animals and in Man

We have induced all four auricular arrhythmias in the dog with an irritable auricle by mechanically teasing the auricular wall. Similarly, by mild stimulation of the human auricle we have produced auricular premature systoles and auricular paroxysmal tachycardia. A counterpart of these experiments is the occurrence of auricular premature systoles,[650] flutter[341] and fibrillation[341] following trauma to the human heart. A variety of arrhythmias may be precipitated by surgical procedures in the heart or lung (Levine).[341]

Intra-cardiac catheterization has been followed by various arrhythmias, including auricular premature systoles, auricular paroxysmal tachycardia and flutter (Figure 312). Such instances are not necessarily due to direct cardiac trauma to the heart as the arrhythmia sometimes occurred before the catheter reached the heart

The fact that each of the auricular arrhythmias can be produced in man and in the dog by mechanical stimulation of the auricular myocardium may be considered as indirect evidence of the fundamental similarity of these disorders.

Observation 11: Clinical Associations and Transitions Between the Arrhythmias

The occurrence of two or more of the auricular arrhythmias in a single patient is a common clinical observation.[20, 276, 458, 587, 669] Persons who suffer from auricular paroxysmal tachycardia or paroxysmal fibrillation often exhibit premature systoles during the quiescent intervals. A given patient may undergo rapid transitions from one rhythm to another or may exhibit each of the arrhythmias at different examinations. In numerous patients with mitral stenosis the gradual evolution of permanent auricular fibrillation from premature systoles

experimentally produced auricular arrhythmias is identical. All observed differences between these disorders are directly related to variations in the rate of discharge from the initiating ectopic focus.

Clinically, the relationship between the auricular arrhythmias may be less clearly defined. The concept of the unitary nature of these disorders is supported by the frequent occurrence of two or more of the arrhythmias in a single patient, occasionally within a few minutes; by certain similarities in their response to quinidine and digitalis; by the occurrence of all four arrhythmias following trauma to the heart; by the basic similarity in the configuration of the auricular deflections of auricular premature systoles, tachycardia and flutter in esophageal, limb and chest lead electrocardiograms; and by the difficulty often encountered by clinicians in differentiating frequent premature systoles from tachycardia, tachycardia with auriculoventricular block from flutter, flutter with ventricular aberration from fibrillation. Finally, the consistent finding that all cinematographic, electrocardiographic and oscillographic features of the auricular arrhythmias demonstrable in human subjects are similar to those observed in the experimental animal provides convincing evidence that the relationship established by experimental methods also exists clinically. It is generally recognized that clinical auricular premature systole and auricular paroxysmal tachycardia are basically similar, this impression is confirmed by our observations of the experimentally produced disorders in human subjects Likewise, the close association between clinical auricular flutter and fibrillation is well known The fact that clinicians have long been on uncertain ground when attempting to differentiate rapid tachycardia from flutter indicates that these two arrhythmias also are fundamentally identical On the other hand, as discussed in Chapter IX, tachycardia and flutter exhibit certain distinguishing clinical features which cannot be fully explained in the light of present knowledge.

The vast clinical experience which has dictated certain distinctions in the behavior and prognosis of the various arrhythmias cannot be challenged. But the assumption that these clinical distinctions necessarily imply the existence of fundamentally different mechanisms is not warranted by the experimental facts. That such apparently diversified conditions as innocuous premature systoles and a fatal tachycardia are merely different forms of the fundamental disturbance may appear improbable; it is not, however, unparalleled. An analogy may be drawn with pulmonary tuberculosis. A calcified Ghon complex and rapidly fatal tuberculosis present widely divergent clinical pictures, *yet their* origin from the tubercle bacillus, and the essential nature of the underlying disturbance is *identical.*

The reduction of four clinically distinct auricular arrhythmias to a common mode of origin helps to clarify a previously confused relationship. Clinical similarities are readily understood in terms of this concept, while hitherto unexplained clinical differences may be correlated with differences in auricular rate. Most important, the elucidation of the mechanism of the arrhythmias places treatment, heretofore empirical, on a rational basis and may well lead to improvements in therapy as further knowledge is obtained concerning the pharmacology of various drugs.

The four auricular arrhythmias produced in experimental animals as described in this monograph differ in *degree rather than* in *nature* of the underlying disturbance. In our opinion, the existence of a similar relationship between the four clinical auricular arrhythmias has been demonstrated. Whether the unitary nature of the clinical auricular arrhythmias is considered as a theory or as an [illegible] upon the judg[illegible] and, ultimately, [illegible] the results of further investigation. We submit this concept in the hope that it will prove of value to medical students, to clinicians and to other research workers concerned with the study and treatment of the auricular arrhythmias.

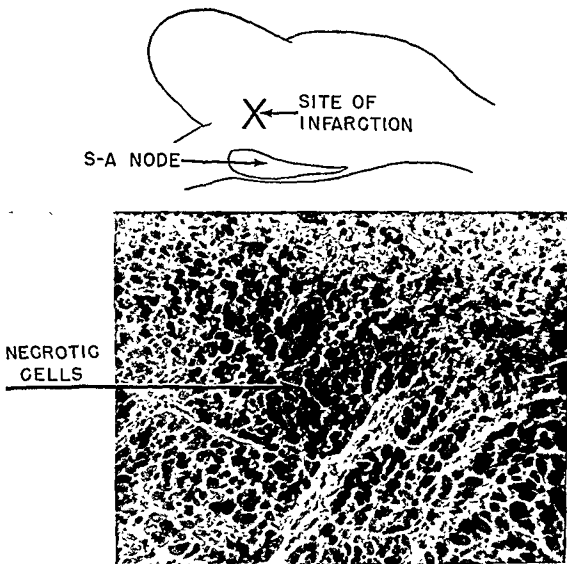


Figure 317. Location of the infarcted area together with histologic appearance of post mortem specimen from patient whose tracings appear in Figure 316 Note acute necrosis of cells due to the auricular infarction The site of maximum infarction was at the base of the right appendix, ventral to the S-A node, thus, note that in Figure 316 the P′ waves are upright and similar to those in normal sinus rhythm

through auricular tachycardia, flutter and paroxysmal fibrillation is observed. Occasionally, three or four arrhythmias are present in a single electrocardiographic record (Figures 313, 314 and 315).

After coronary occlusion with auricular infarction we have frequently observed many of the auricular arrhythmias in rapid succession (Figures 316 and 317) It is unlikely that disorders *having radically different* mechanisms should occur in a single heart in the course of a few moments.

DISCUSSION

The cinematographic, electrocardiographic and oscillographic observations made during the course of our study of the auricular arrhythmis reveal the answers to many questions concerning the relationship between these disorders. Experimentally, any or all of the arrhythmias may be initiated by *either* electrical, chemical or mechanical stimulation which sets up a discharging ectopic focus at a given site on the auricles. Regardless of the method of production, the rate of discharge of stimuli from the focus determines which arrhythmia occurs. Excision of the focus converts any of the arrhythmias to normal sinus rhythm. In our opinion, this series of observations establishes beyond question that the mode of origin of the

experimentally produced auricular arrhythmias is identical. All observed differences between these disorders are directly related to variations in the rate of discharge from the initiating ectopic focus.

Clinically, the relationship between the auricular arrhythmias may be less clearly defined. The concept of the unitary nature of these disorders is supported by the frequent occurrence of two or more of the arrhythmias in a single patient, occasionally within a few minutes; by certain similarities in their response to quinidine and digitalis; by the occurrence of all four arrhythmias following trauma to the heart; by the basic similarity in the configuration of the auricular deflections of auricular premature systoles, tachycardia and flutter in esophageal, limb and chest lead electrocardiograms; and by the difficulty often encountered by clinicians in differentiating frequent premature systoles from tachycardia, tachycardia with auriculoventricular block from flutter, flutter with ventricular aberration from fibrillation Finally, the consistent finding that all cinematographic, electrocardiographic and oscillographic features of the auricular arrhythmias demonstrable in human subjects are similar to those observed in the experimental animal provides convincing evidence that the relationship established by experimental methods also exists clinically. It is generally recognized that clinical auricular premature systole and auricular paroxysmal tachycardia are basically similar, this impression is confirmed by our observations of the experimentally produced disorders in human subjects Likewise, the close association between clinical auricular flutter and fibrillation is well known The fact that clinicians have long been on uncertain ground when attempting to differentiate rapid tachycardia from flutter indicates that these two arrhythmias also are fundamentally identical. On the other hand, as discussed in Chapter IX, tachycardia and flutter exhibit certain distinguishing clinical features which cannot be fully explained in the light of present knowledge

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[illegible] the results of further investigation. We submit this concept in the hope that it will prove of value to medical students, to clinicians and to other research workers concerned with the study and treatment of the auricular arrhythmias.

Methods and Equipment

All scientific research, regardless of the inherent difficulties of the problem under investigation, is qualified by the capacity and accuracy of the equipment available. In the study reported in this monograph the nature of the auricular arrhythmias has been examined with new as well as previously applied experimental methods and equipment. Electrocardiographic, cathode-ray oscillographic and high speed cinematographic techniques were employed in both man and animals, and the results correlated whenever possible. The present section consists of (1) a summary of the methods of study on humans and dogs; and (2) a detailed description of the specific apparatus, some of which is new to the field of cardiology.

METHODS OF STUDY ON MAN

SPONTANEOUS ARRHYTHMIAS IN MAN

The auricular arrhythmias – premature systoles, paroxysmal tachycardia, flutter and fibrillation – were studied in human subjects exhibiting the spontaneously arising disorders. In each instance simultaneous multiple electrocardiograms were recorded using various combinations of standard, unipolar limb and precordial leads. Many of the subjects also were examined by means of simultaneous esophageal leads from several auricular levels. In addition, limb and esophageal leads from patients with auricular flutter and fibrillation were recorded on the dual-beam cathode-ray oscillograph. High-speed cinematographs of the left auricle were taken simultaneously with electrocardiograms in two instances of auricular fibrillation. The electrocardiographic, oscillographic and cinematographic equipment used in these studies is described later in this section.

In all unipolar lead electrocardiograms the indifferent electrode was Wilson's central terminal.[661, 662, 663, 664, 665]

Simultaneous esophageal lead electrocardiograms were obtained from high, middle and low auricular levels; in most instances one unipolar limb lead was also recorded and the pattern correlated with that in the esophageal lead tracings. When records from the esophagus were made on the dual-beam cathode-ray oscillograph, either two esophageal leads or one unipolar limb lead and one esophageal lead were recorded simultaneously. In several patients auricular intrinsicoid deflections in precordial leads were so prominent that the course of the excitation wave of the arrhythmia could be charted by taking numerous leads from adjacent areas over the entire chest.

EXPERIMENTALLY PRODUCED ARRHYTHMIAS IN MAN

Auricular premature beats and paroxysmal tachycardia were produced experimentally in 18 human subjects during surgical procedures in the thorax (Chapter IV). The pericardium was opened and the auricules stimulated under direct vision in 13 of the patients. Because of pulmonary tuberculosis or other danger of contamination, the pericardial cavity was not opened in the remaining five subjects; in these instances various parts of the auricles were readily identified through the thin semi-transparent pericardium and stimulated without difficulty. Stimulation consisted of gentle

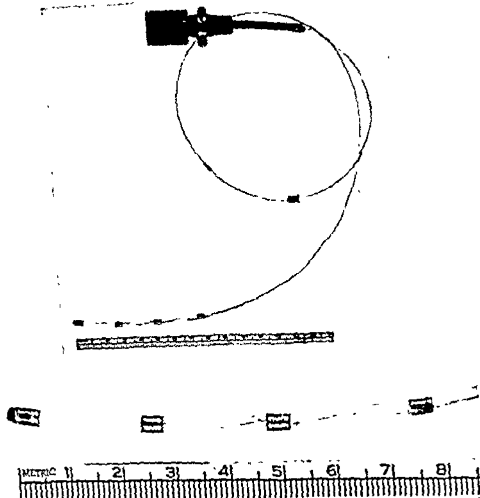


Figure 318 Esophageal lead used to record four simultaneous electrocardiograms.

Upper figure full length esophageal lead with four electrodes attached. This tube can be readily swallowed or inserted through the nose so that the electrodes lie directly behind the auricles Position of the electrodes in the esophagus is determined by observing the configuration of the auricular and ventricular deflections recorded in the esophageal lead electrocardiograms

Lower figure lower end of the esophageal lead approximately 2 X normal size, showing the four contact rings 2.5 cm. apart connected by separate wires to their terminals through a plastic (polyethelene) tube

pinching of the auricle or irritation with a blunt forceps In each instance records were made on the multiple channel electrocardiograph from standard limb leads Three esophageal leads were recorded simultaneously with one limb lead (usually lead 2, 3 or AVF) in 10 subjects, by this method the auricular patterns in limb and esophageal leads could be correlated. On two occasions high speed cinematographs of the right auricle were recorded during the experimentally produced arrhythmias.

ESOPHAGEAL ELECTRODE

The esophageal electrode used in electrocardiographic and oscillographic studies of the spontaneous arrhythmias in man is illustrated in Figure 318.[471] Four electrodes each 3 x 5 millimeters in size were fastened 2.5 centimeters apart on a polyethylene catheter 0.168 centimeter in outside diameter. Because of the possibility of depolarization currents with brass, some of the electrodes were constructed of nickel silver; however, no variations attributable

to the difference in material of the recording electrode were demonstrable in the tracings.[471] Insulated wires (Litz No. 8817) connected each electrode tip to a separate binding post. As many as four records, from leads spanning almost the entire length of the auricles could be made simultaneously. The electrodes were swallowed with little difficulty by most patients and retained as long as necessary, usually for approximately an hour. The most satisfactory records were obtained if the subject gripped the catheter gently between his teeth and held his breath while the tracings were made.

The esophageal electrodes used in the study of experimentally produced arrhythmias in man consisted of nickel silver rings fastened 5 centimeters apart on a Levine tube. Each electrode was connected independently by insulated wires to a separate binding post.

ANALYSIS OF ELECTROCARDIOGRAMS IN MAN

Through the courtesy of the Los Angeles County General Hospital, the Cedars of Lebanon Hospital, and several physicians in private practice, we had access to at least 100,000 electrocardiograms made during the past 20 years. From this collection over 500 electrocardiograms of auricular arrhythmias were selected for detailed examination. These records provided material for the analysis of electrocardiograms of auricular tachycardia and flutter in man presented in Chapter IX, for the various examples of arrhythmias shown in Chapter X, for some of the observations on the site of origin of auricular fibrillation in Chapter XIV, and for miscellaneous illustrations throughout the text.

METHODS OF STUDY ON DOGS

Auricular arrhythmias were experimentally produced in over 300 dogs' hearts and were studied by electrocardiographic, cathode-ray oscillographic and cinematographic techniques. Whenever obtainable, large animals weighing 20 to 25 kilograms or more were used. In earlier experiments the animals were anesthetized with intravenous sodium pentobarbital in dosage of 30 milligrams per kilogram of body weight; supplementary sodium pentobarbital was administered as needed. During the course of the study we found that morphine followed by intravenous urethane (25% solution) was an excellent anesthetic and did not cause a rapid heart rate. Artificial respiration was carried out through an intratracheal cannula inserted at the onset of the experiment. A piston-type automatic respirator was employed which pumped 100 per cent oxygen at a constant flow of about seven liters per minute.

OPERATIVE TECHNIQUE

The operative technique for cardiac experiments generally employed in physiology laboratories permits only limited exposure of both auricles. In order to achieve wider exposure, especially of the body of the left auricle, the following technique of dissection was developed and used in our study. Little time is consumed in exposing the heart (approximately 10 minutes) and shock is minimal during the experimental procedure. This method is recommended as a useful, relatively non-traumatizing exposure for experiments on the heart and lungs.

A transverse incision is made over the fourth rib from one axilla to the other. The third, fourth and fifth ribs are exposed and removed subperiosteally on both sides as far back as the posterior axillary line. The subperiosteal technique is used to avoid bleeding. The internal mammary vessels are ligated and divided; the sternum is split transversely at the level of the fourth intercostal space. The pleurae in the fourth intercostal spaces are opened bilaterally; artificial respiration is started and adjusted at this point. The cut ends of the sternum are now widely retracted, the heart and lungs are exposed. A cruciate incision is made in the pericardium. The transverse component of this incision is made slightly cephalad to the point of entry of the inferior vena cava into the right auricle and extends as far dorsally as possible



Figure 319 Photograph of right side of heart demonstrating the surgical incision which gives a wide exposure of the heart From this view of the heart the right ventricle, the whole of the right auricle, the superior and inferior venae cavae are exposed.



Figure 320 Photograph of left side of heart demonstrating exposure of the appendix and body of the left auricle, pulmonary *veins, etc* Note how this exposure reveals full detail of the body of the left auricle, the left auricular appendix and pulmonary veins.

on the two sides of the heart. The longitudinal component is made in the midline from the diaphragmatic surface of the heart to the pericardial reflection on the aorta. The cut edges of the pericardium are stretched over the lungs and are sutured to muscles deep in the axillae; thus the auricles, the ventricles and the venae cavae are widely exposed. By tilting the board on which the animal is resting to one side or the other, the desired auricle may be brought into view. This exposure is also admirably suited for electrocardiographic or oscillographic recordings of direct auricular leads. An intravenous infusion of normal saline solution is usually started into a femoral vein and continued throughout the experiment at a rate of one to two cubic centimeters per minute.

During experiments in which a small portion of the auricle is photographed at a short distance (8 to 10 inches) from the camera, further dissection is necessary to obtain a clear field and adequate lighting of the area under study. In such instances the entire lateral thoracic wall is resected for a distance of three to five inches cephalad and caudad to the auricle to be photographed.

Figures 319 and 320 illustrate the exposure of the right and left sides of the heart obtained with the dissection technique described above.

METHODS OF PRODUCING ARRHYTHMIAS

Auricular arrhythmias were produced in dogs by three methods: (1) electrical stimulation; (2) aconitine application; and (3) mechanical stimulation.

(1) Electrical stimuli were applied through a fine copper-wire electrode stitched to the auricle at the location selected as the ectopic focus. Shocks at rates of 50 per minute to 1,000 per minute were introduced by means of a pulse generator. The frequency of electrical stimulation determined the type of arrhythmia produced. Although the threshold levels varied considerably in different animals, in most instances 50 to 100 shocks per minute generated premature systoles; rates of 150 to 300 per minute instituted the rhythm of auricular tachycardia; 300 to 400 stimuli per minute produced the characteristics of auricular flutter. The rate of stimulation required for the production of auricular fibrillation varied from animal to animal. Fibrillation was seldom produced at stimulatory rates below 400 per minute, whereas in some animals stimulation at rates as high as 600 per minute was required. In other animals the auricles would not respond at rates rapid enough for the production of fibrillation because of auricular blocking.

When flutter was produced by electrical stimulation of the auricles at suitable rates over a period of several seconds, the arrhythmia frequently continued for seconds or even many minutes after the stimulation was terminated. A similar phenomenon was observed when auricular fibrillation was instituted by electrical stimuli. Early in this series of experiments, only that phase of an arrhythmia which persisted after stimulation had been discontinued (post-stimulatory arrhythmia) was considered suitable for study. We soon noted that the arrhythmia occurring during the electrical stimulation was cinematographically identical with that occurring as an after-effect. Consequently, in later experiments films were made during as well as after the period of electrical stimulation. The experimentally produced arrhythmia could thus be maintained for photographic study for a prolonged period. Electrocardiographic records taken during the period of stimulation were disfigured by adventitious deflections due to the stimuli themselves.

(2) Aconitine was first used by Matthews[443] and Cushny.[123] Scherf[542] injected aconitine into the sino-auricular node and applied crystals to the auricular surface. Since both of these methods proved difficult to standardize, the following technique was developed and used in our laboratory. An 0.05 to 5.0 per cent solution of aconitine in benzene was employed to obtain auricular fibrillation. Since the dissolved drug became inactive on standing, a fresh solution was prepared each day. The selected area on the wall of the auricles was carefully dried. A

cotton swab one millimeter in diameter on the end of a thin applicator stick was soaked with aconitine solution, applied to the dried portion of the auricle, and held in position for about 60 seconds. By employing a small swab the ectopic focus could be sharply localized. Auricular fibrillation often began one to two minutes after the aconitine-soaked swab was removed, usually preceded by a few premature systoles, a short run of auricular tachycardia, or even by auricular flutter. In most instances one application of aconitine was sufficient, occasionally, especially in young animals, several applications were necessary before auricular fibrillation or flutter appeared. Care was taken to avoid touching the ventricles with the aconitine swab lest a fatal ventricular arrhythmia be produced.

In early experiments only the lower concentrations of aconitine were employed. These concentrations proved satisfactory for the production of the slower rate arrhythmias, namely, auricular premature systole or paroxysmal tachycardia. To produce auricular flutter or fibrillation, however, repeated applications of the lower concentrations were necessary and frequently the solution unavoidably spread over a larger surface of the auricle. If a ventricle came in contact with the drug, ventricular fibrillation usually occurred and the experiment terminated prematurely. Consequently, in later experiments single applications of the higher concentration were used to obtain the more rapid rate arrhythmias. When 5.0 per cent aconitine was applied by the method described, auricular fibrillation usually ensued within a minute and persisted for the duration of the experiment. Since the stronger solution could be applied to a restricted area, the danger of ventricular contamination was decreased.

After auricular fibrillation was established by aconitine, any desired auricular arrhythmia usually could be produced by the following modification of the cooling technique first developed by MacWilliam in 1887.[653] The site of aconitine application was cooled by direct application of an ethyl-chloride spray or a block of ice. Shortly after the cooling agent was applied, cardiac standstill and/or normal sinus rhythm resulted. If the cooling process was stopped and the aconitine site allowed to thaw, the cardiac rhythm often repeated the series of changes observed after the application of aconitine, namely (from auricular standstill) to sinus rhythm, to auricular premature beats, to auricular tachycardia, to auricular flutter, and finally to auricular fibrillation. The change in rhythm with cessation of cooling did not follow an invariable pattern in all animals or in one animal subjected to repeated experiments. Often one or more of the arrhythmias failed to develop during the progression from cardiac standstill to auricular fibrillation. In the vast majority of experiments, however, by using the proper concentration of aconitine and by judicious cooling, transitions between any of the arrhythmias could be produced and each arrhythmia maintained for a period long enough to permit both electrocardiographic and cinematographic study. The details of these transitions and their clinical implications are discussed in previous chapters.

(3) In a few experiments, auricular premature beats, tachycardia, flutter and fibrillation were produced by mechanical teasing of the auricles with a wooden applicator or by heating the auricle with hot lamps. These methods were not considered reliable and were rarely used.

In our experience, the local application of aconitine was by far the most effective method for the production of the auricular arrhythmias. Each disorder could be produced more simply and with more certainty than when other experimental methods were employed.

INVESTIGATIVE TECHNIQUES

The four experimentally produced [illegible]

[illegible] experiments recorded by high-speed cinematographs; a film was included for study only if the pattern obtained in the tracing was characteristic of a given arrhythmia. Over 100,000 feet of film depicting experiment-

ally produced auricular arrhythmias in more than 120 dogs were studied. Every example of a given arrhythmia was cinematographically identical whether produced by aconitine, electrical or mechanical stimulation.

Direct auricular and unipolar limb lead electrocardiograms were made with the four-channel electrocardiograph. Additional direct auricular and unipolar limb lead tracings of auricular fibrillation were made with the Sanborn Twin Beam Electrocardiograph. The electrical manifestations of the arrhythmias in more than 45 animals were recorded on the dual-beam cathode-ray oscillograph. The same electrodes and connections employed in electrocardiographic studies were used to obtain oscillograms except that three or four leads could be recorded simultaneously with the electrocardiograph and only two with the oscillograph. In each instance the exploring electrode was paired with an indifferent electrode connected to the three limbs according to the unipolar method described by Wilson.[605] Curare was administered to 10 dogs under oscillographic study in an attempt to eliminate fine potentials due to muscle tremor; the drug had no apparent effect on the heartbeat and the tracings were comparable in all respects to those from animals given no curare.

DIRECT LEAD ELECTRODES

In animal experiments designed to trace the course of the electrical impulse in the auricle, non-polarizable soft-tipped electrodes similar to those described by Lewis [385] were used (Figure 321). Each electrode consisted of a small glass tube tapered at its lower end which was connected to a short length of soft rubber tubing. The tip of the glass tube was plugged with a slated kaolin wick and was filled above the plug with 10 per cent copper chloride solution in which a long coil of fine copper wire was immersed. The short rubber extension was packed with cotton-wool saturated with isotonic saline solution. When pressed lightly against the epicardium, this soft-tipped electrode caused little or no "injury current." For experiments designed to determine the speed as well as the direction of the electrical impulse, special holders were devised to separate the electrodes at a constant and known distance (Figure 321). In certain special experiments other electrodes were used; these included steel spring clip-on electrodes, silver-silver chloride electrodes and platinum electrodes.

The auricular deflections in the direct lead tracings usually could be identified with certainty. They consisted of short monophasic or diphasic deflections of relatively large amplitude. The intrinsic deflection was defined as the onset of the wave of negativity in each complex;[385] this deflection generally is considered to represent the time of arrival of the impulse beneath the electrode. The small or minor deflections preceding and following the intrinsic deflection were assumed to represent the passage of the impulse through neighboring or distant masses of cardiac muscle and constitute extrinsic deflections.

Lewis carried his measurements and calculations to four significant figures[385] in timing his electrocardiographic experiments. We found such close measurements of electrocardiograms often incorrect and therefore utilized only two significant figures. Because of the magnification in amplitude of the deflection and the rapid recording rates achieved with the cathode-ray oscillograph, measurements made from oscillographic tracings were more accurate and could be carried to three or more significant figures.

EQUIPMENT

ELECTROCARDIOGRAPH

The electrocardiographic equipment used to study the auricular arrhythmias in both man and dogs was the Technicon Cardiograph* and the Sanborn Poly-Viso Recorder. Three leads were recorded simultaneously on the former; four on the latter. These machines afforded great flexibility in our experiments. We were able to use any combinations of standard, unipolar limb leads, precordial leads and esopha-

* Kindly loaned by the Technicon Corporation.

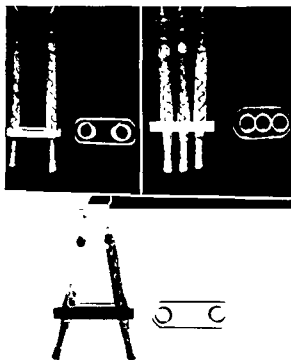


Figure 321 Illustration of paired electrodes Three typical arrangements of non-polarizable copper-copper sulfate electrodes with soft-tipped boots Each electrode consists of small glass tube tapered at the lower end, containing 10 per cent copper sulfate solution and coiled copper wire. The tapered tip is plugged with Kaolin wick [illegible]

[illegible] trodes at a fixed distance apart

geal leads. Either machine was operated at standard (25 millimeters per second) or double (50 millimeters) paper speeds

A Sanborn Twin Beam Electrocardiograph* was used to record direct auricular and unipolar limb lead tracings of auricular fibrillation in one experiment (Figures 237 and 238). This instrument is a photographic recorder The galvanometers have a natural frequency of 500 cycles per second, critically damped, and a deflection speed of 1 millisecond (0.001 second). The records were made at paper speeds of 25 to 75 millimeters per second

CATHODE-RAY OSCILLOGRAPH

Early in our study of auricular fibrillation we found that the value of standard mechanical electrocardiographic equipment was limited by several factors: (1) insufficient amplification; (2) mechanical inertia; (3) inability of the machine to respond accurately at frequencies higher than 50 cycles per second; and (4) restriction of paper speed to 25 to 50 millimeters per second. To overcome these limitations the cathode-ray oscillograph[601] was used.[155, 442, 497, 503, 504, 525] This electronic apparatus provided sufficient amplification for purposes of study. A useful magnification several thousand times greater than that in the ordinary electrocardiogram can be obtained. No moving parts are present to introduce inertia, and the frequency response is virtually unlimited. The maximum recording film speed of the Du Mont Oscillograph-Record Camera is 1,500 millimeters per second or 30 times the recording speed of the standard electrocardiograph.

Because the oscillograph cannot record unamplified cardiac potentials it must be used in conjunction with a pre-amplifying device. An amplifier made by staff members of the California Institute of Technology was employed early in the study; this was later replaced by two Grass P 4 pre-amplifiers which had higher frequency responses more suitable for the study of the auricular fibrillation Leads from the subjects were connected to the two pre-amplifiers which in turn were connected to the Du Mont Type 279 Dual-Beam Cathode-ray Oscillograph. This instrument consists essentially of two oscillographs with a single cathode-ray tube 12.5 centimeters in diameter. The tube contains two identical, ceramically separated electron guns which discharge electron beams against the fluorescent tube front. With this apparatus, any two leads may be viewed simultaneously.

At the amplification range used in our experiments, square waves tests showed the response to be linear both in the pre-amplifiers and in the oscillograph.

In order to avoid electrostatic alternating current (a.c.) artefacts, a copper wire-screen cage was constructed to enclose the recumbent subject being examined The cage was large

* We wish to thank Mr M Rappaport for the use of his time and his machine in one of these experiments

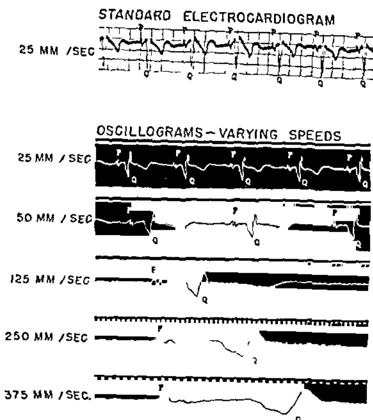


Figure 322. Esophageal lead in a normal patient recorded from mid-auricular level.

Upper illustration from esophageal lead recorded on standard electrocardiograph.

Lower illustration, same esophageal lead recorded by oscillograph The recording speeds have been varied from 25 mm. to 375 mm. per second without increase in vertical amplitude, to demonstrate the effect of recording speed on configuration of auricular and ventricular complexes

enough to accommodate both subject and laboratory personnel during experiments on man or animals.

Two alternative techniques were used to photograph the oscillograph tube front: (1) continuous strip film recording; and (2) direct cinematography

Continuous strip-film recording: The output from the pre-amplifiers was to the X (horizontal) axis of the oscillograph; the electron beam appeared as a dot which moved back and forth horizontally in a single line across the tube face When the film was pulled vertically across the tube front at a constant speed, the moving dot was recorded as a continuous line comparable to a standard electrocardiogram. This technique was accomplished with the electronically controlled Du Mont Type 314-A Oscillograph Record Camera which photographed through a light-tight periscope attached to the oscillograph cabinet. The speed with which the film was pulled through the camera constituted the time-base of the finished record. The film speed could be varied from 2.5 centimeters per minute to 150 centimeters per second (Figure 322). Most records were made at speeds of 12.5 to 37.5 centimeters per second, that is, 5 to 15 times the recording speed of the normal electrocardiograph. The cardiac potential could be amplified by the oscillograph to produce a deflection which more than covered the horizontal diameter of the tube. Thus, by synchronous adjustment of the amplitude (amplification) and time-base (film speed), a given deflection could be magnified in horizontal and vertical dimensions to about 500 times its standard electrocardiographic size.

Direct cinematography of oscillograph tube front: Like the electrocardiograph and the Du Mont Oscillograph Record Camera, this recording technique yields a pattern with both amplitude and a time-base. The output from the pre-amplifiers was to the Y (vertical) axis; the patterns traced by the moving electron beams were recorded with the specially adapted Bell and Howell Camera at 100 to 200 frames per second and by the Western Electric Fastax Camera at 500 to 2,000 frames per second. The sweep speed of the oscillograph was adjusted separately for each film speed and for each arrhythmia. That is, it was necessary to increase the number of times per second the electron beam moved across the tube front in order to (1) obtain satisfactory photographs at higher camera speeds, and (2) observe the finer details of the rapid rate arrhythmias. In general, most satisfactory records of auricular fibrillation in man were obtained at film speeds of 750 to 1,000 frames per second with about 10 sweeps per second. These photographs were made on Eastman Super XX film which was specially developed to bring out the faint images inscribed at high recording rates.

The magnification achieved by this technique is many times greater than that obtained with continuous strip-film photography. Four kinds

of magnification are utilized in the cinematographic recording technique. (1) As in the ordinary electrocardiograph, the pre-amplifier magnifies the impulse to within the range of sensitivity of the oscillograph (2) The oscillograph magnifies the amplified impulse many times For example, an impulse of 0.5 millivolts can be made to exceed the vertical screen diameter of 12 5 centimeters. Proper adjustment of the horizontal axis of the oscillograph permits similar magnification in a horizontal plane. (3) By means of high speed photography, magnification in time may be accomplished to such a degree that the events of one cardiac cycle require a projection time of 6 minutes. In addition, by adjustment of the speed with which the electron beam sweeps across the tube front, the horizontal distance traveled by the beam during one cycle may be increased many times. For example, if the sweep speed is 2 per second, the electron beam will travel a horizontal distance equal to twice the diameter of the tube front, or 25 cm in one second. If the sweep speed is now increased 10 times, the distance which the beam travels in one second will be 10 times the 12 5 centimeters diameter of the tube, or 125 centimeters. (4) Final magnification is achieved in projection This last step depends on the resolving power of the camera and projector lenses, the sensitivity and grain of the film, the light source in the projector, and the age and type of screen used By proper selection of oscillograph settings, camera, films, developer, projector and screen, an estimated useful final magnification of about 5,000 times normal electrocardiograph size may be obtained

Because of technical differences between the two types of photography described above, two distinct types of cathode-ray tubes were employed For continuous strip-film photography a tube with short-persistent phosphorescent qualities (the Du Mont 5SP11) was used Photographs of this tube front showed only the moving fluorescent dot produced by the electron beam. For high-speed cinematography (100 to 2,000 frames per second), a tube was employed which had relatively long-persistent phosphorescence (the Du Mont 5SP7). This tube yielded a photograph similar to the electrocardiogram in appearance. Instead of a moving dot, complete wave patterns were recorded which slowly faded out as the phosphorescence decreased in intensity.

HIGH SPEED CINEMATOGRAPHY

Auricular contractions during the arrhythmias occur with such rapidity that they cannot be studied with the unaided eye.[79, 219, 243, 326, 396, 592, 593, 647] By means of high-speed cinematographs of the intact heart, the muscular movements of the auricles may be visualized at rates slow enough to permit accurate observation and the image magnified so that the finest details of the contractions are revealed. This technique was employed extensively during our experimental study of the auricular arrhythmias in dogs. Relatively fewer cinematographs were taken in human subjects. Identical apparatus and photographic methods were used in man and animals except for minor modifications designed to eliminate possible adverse effects on the patients.

In order to obtain photographs which are suitable for study, three major factors must be carefully considered. These are: (1) the camera, (2) lighting; and (3) projection of the picture.

Cameras: Two types of cameras were employed in the study. A specially adapted Bell and Howell 16 millimeter Specialist camera was used to take pictures at 16, 100 and 200 frames per second. Motion pictures at rates between 500 and 3,000 frames per second were taken with a 16 millimeter Western Electric Fastax camera with an F 2.3 Astro lens. High speed cinematographs could be recorded with the Fastax camera in minimal exposure times; the significance of this fact is pointed out in the discussion of lighting. The heart was photographed at various camera speeds depending upon the specific auricular movements selected for study. Gross movements of the auricles were seen best on films taken at 100 frames per sec-

ond (approximately six times slower than normal motion). At this speed the observer could become oriented to gross events as they occurred in the cardiac cycle and was better able to understand their relationships than on cinematographs taken at faster speeds. The site of origin and speed of the contraction wave, the intensity of contiaction, the duration of systole and diastole, and the effect of certain drugs on these characteristics were observed most clearly on films exposed at 500 to 1,000 frames per second (approximately 30 to 60 times slower than normal motion). Auricular flutter (tachycardia of the flutter type) and auricular fibrillation were studied most successfully on films exposed at 750 to 3,000 frames per second (approximately 45 to 185 times slower than normal motion).

The majority of cinematographs of the auricles were taken with the camera about 1 meter away from the heart. For a special high magnification study of a segment of the auricle approximately 1 square centimeter, the lens was equipped with extension tubes and the distance from the camera to the areas under observation was 20 to 25 centimeters.

The depth of the field photographed was increased by decreasing the shutter aperture; diaphragm scale settings of f 6.3 to f 12. were usually used. By this device the auricular structures were maintained in sharp focus throughout the cardiac cycle, except during the high magnification study when the small segment of auricle could not be kept in focus in all cases due to the vigorously pulsating ventricles. If possible, the camera was re-framed and refocused after every roll of film was exposed, in no instance were more than three rolls of film exposed without this precaution. The area to be photographed was parallel to the surface of the lens when feasible, so that sharp focus could be obtained. All photographs of the auricles were made on Type A Kodachrome Ciné Kodak film.

Lighting: All high-speed motion pictures must be taken in a bright light. As a general rule, the faster the photographic speed, the brighter must be the illumination. At camera speeds from 16 to 200 frames per second, two or three photospot lamps were needed. When pictures were taken at faster speeds, 500 to 3,000 frames per second, additional light was required. General Electric 750-R photospot lamps in banks of four lights each were placed in horseshoe fashion around the camera. This type of lamp has a focal distance of 45 centimeters. The lamps were held in an incandescent type B-6 frame made by Bardwell and McAlister, Inc. From 12 to 20 lamps were adjusted in this fashion and focused individually so that the beams were concentrated in an area of approximately 100 square centimeters or less. The amount of illumination on the heart when 12 lights were used was estimated at approximately 700,000 foot-candles, when 20 lights were used the intensity was about 1,200,200 foot-candles. By contrast, sunlight on the earth at noon on a clear day equals about 11,000 foot-candles.

The heat generated by the lamps, especially when 20 were used, was so intense that it frequently caused a speeding of the heart rate and drying of the exposed tissue after two to four seconds; on several occasions the auricle being studied was actually burned. In some instances spontaneous arrhythmias followed the prolonged use of the lights; in others, auricular standstill occurred and persisted until the intense heat was removed. This problem of heating was solved by various methods. As noted in the discussion of cameras, high-speed pictures taken with the Fastax camera could be recorded within a fraction of the time required to expose the same footage of film at slower camera speeds For example, a roll of 50 feet (15.24 meters) of film was exposed in four seconds at camera speeds of 500 frames per second; the same footage was exposed in two seconds at 1,000 frames per second, in one second at 2,000 frames per second, and in ½ second at 3,000 frames per second. Since the photospot lamps were turned on only for the duration of the filming, the heart was never exposed to the intense heat for more than a second when fast camera speeds were used. When lower speed pictures (500 to 1,000 frames per second) were

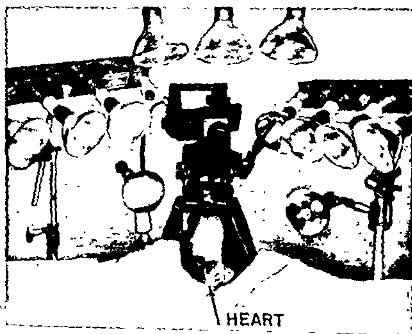


Figure 323 Typical set-up for high speed motion picture photography of exposed dog's heart demonstrating Western-Electric Fastax camera, banks of photospot lamps, draped animal and exposed heart.

made, either a maximum of 50 feet of film was exposed or, alternatively, 100 feet of film was used and the heart was bathed with normal saline solution during the last half of the exposure. During experiments in which a large number of lamps (15 to 20) were employed, all exposed parts of the heart except that being photographed were covered with cotton soaked in normal saline solution, this device afforded excellent protection. The mechanism of the Fastax camera is such that a short interval of time is needed for the motors to achieve full speed, especially at recording rates from 2,000 to 3,000 frames per second To avoid unnecessary heating of the auricles, the camera was always started shortly before the lights were turned on. With this procedure the pictures were taken only when the camera was running at the desired speed and the lamps were burning only during the actual photographing time

Despite the precautions used, in most instances the heat from the lamps was sufficiently intense to cause an increase in the heart rate during the last half of each exposure. In order to exclude arrhythmias which may have been altered by the heat, only the first half or two-thirds of each roll of film was used in the study.

Figure 323 illustrates the arrangement of camera, light source, and subject.

Projection of the Pictures: Only projectors with excellent optical systems were used to visualize the motion pictures. A light source of at least 750 to 1,000 watts was found satisfactory. The normal speed of the projectors was 16 frames per second; film exposed at 3,000 frames per second and projected at this speed showed the activity of the auricles approximately 180 times slower than their actual rate. By slowing the projection speed to 8 frames per second, the auricular activity could be slowed to 360 times its actual speed *Hence, when exposures were made at 3,000 frames per second and projected at 8 frames per second, events that actually occurred in the auricle in one second required six minutes to be viewed on the screen.*

Comparison of Films: The most accurate method for comparing two films was to view them simultaneously. This was accomplished by projecting the films simultaneously from two projectors run at the same speed and projected on a single screen so that the two images were adjacent to each other. Projectors with similar

optical systems were used and the films were adjusted to show comparable phases of the cardiac cycle simultaneously. By this method small differences in the appearance and action of the auricles were discovered. For example, if the effect of a cardiac glycoside on the normal auricle was to be studied, the auricle was first photographed under normal conditions. The drug was then administered slowly parenterally. After the drug had exerted its pharmacologic effects, as determined from the electrocardiogram, a second cinematograph was taken in *exactly the same manner as the control film*. The two films were then developed and projected simultaneously, as described above. Minute details of change in color, size, appearance and speed of the contraction wave were dramatically revealed.

A second method for comparing two films consists of "double printing" the two negatives on the same positive film. The location and lens adjustment of the camera, the lights and the position of the subject photographed must remain constant while the two films are recorded. For example, if electrical events are photographed from the oscillograph tube front by the Fastax camera as described earlier, during normal sinus rhythm the electron beam may be directed against the upper half of the oscillograph tube front and photographed at a given speed. An arrhythmia is then produced, the electron beam is directed against the lower half of the tube front and photographed in an identical manner as the control. The two negatives are then printed superimposed on one another. When the finished print is projected, the events occurring during normal sinus rhythm and the experimentally produced arrhythmia appear simultaneously on the same film. This technique is a useful tool in demonstrating differences between rhythms, the actions of drugs, and other effects of specific procedures.

A third method for comparing two films is provided by a specialized process called "optical printing." As in the second method, the conditions under which the two films are recorded *must be identical*. Photographs of a structure are taken before and after a specific procedure and the two films are printed on another film, one above or beside the other. Unnecessary portions of each frame may be omitted in order to emphasize a given part of the structure, such as the auricular appendix. Slight reduction in size of the image on the film may or may not be necessary. This, again, is a useful method for demonstrating the effects of a procedure on a given subject. Like the second method, it is valuable for teaching purposes.

The importance of speed in performing the experiments should be emphasized. Laboratory personnel thoroughly familiar with the procedures were essential to obtain the maximum value from each experiment. The dissection was usually complete within 10 to 15 minutes; the first pictures, either of the heart or of the oscillograph, were taken within another 5 or 10 minutes. If any appreciable delay occurred, heart failure or other changes such as an increase in the heart rate frequently intervened. As a result of "good teamwork," most experiments were complete within an hour from the time the anesthetic was administered to the animal.

If meticulous care and attention were given to all details – surgery, anesthesia, size and position of the animal, camera speed, focusing of the lens, lighting, quality and development of film, and finally, careful projection on a glass-beaded screen in a dark room, truly artistic photographs were obtained. These cinematographs revealed the motion of the auricles in fine detail and clarity, thus serving as a valuable scientific instrument.

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This Book

THE AURICULAR ARRHYTHMIAS

was set, printed, and bound by the Pantagraph Printing and Stationery Company of Bloomington, Illinois. The color engravings were made by the Brown & Caldwell Company of Los Angeles, California. The black and white engravings were made by the Capitol Engraving Company of Springfield, Illinois. The page trim size is 8½ x 11 inches. The type page is 39 x 51 picas. The type face is Linotype Caledonia, set 11 point on 13 point. The text paper is 80-pound Warren's Lustro Enamel. The cover is Bancroft Buckram 2120.

